


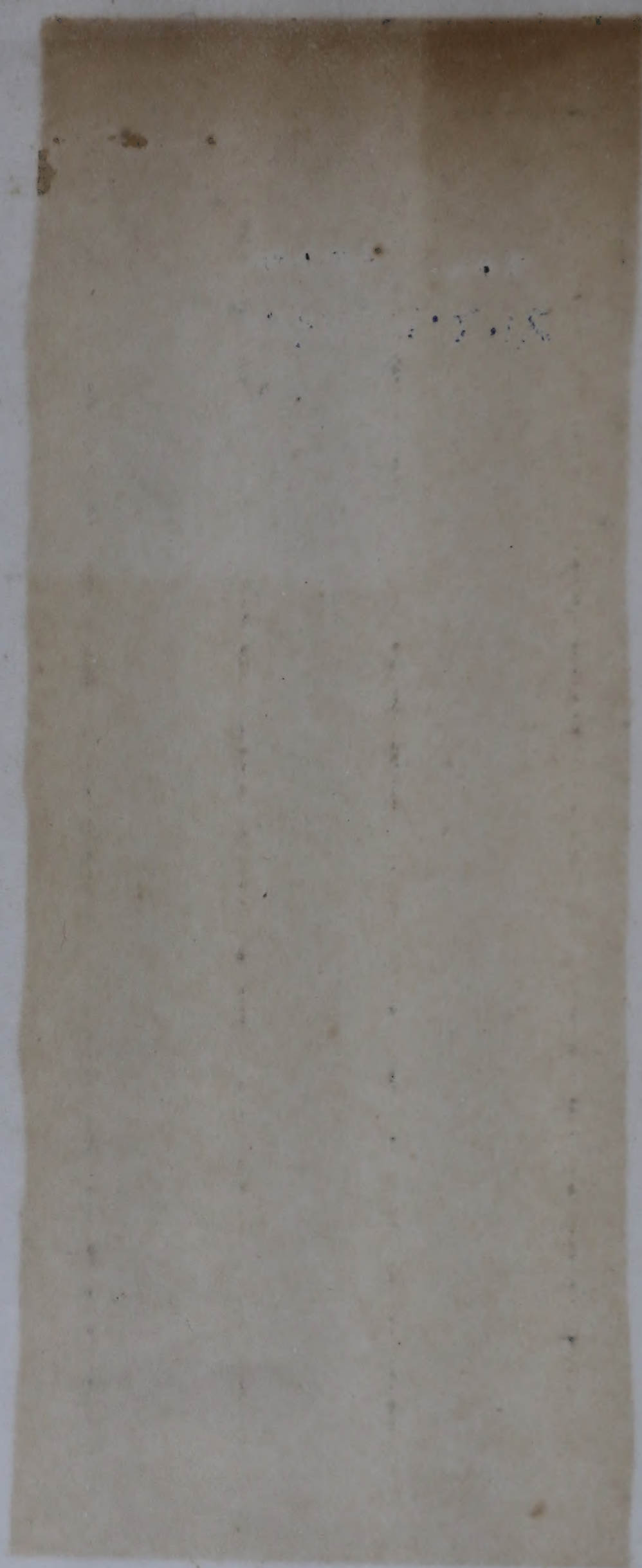
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Biomicroscopy of





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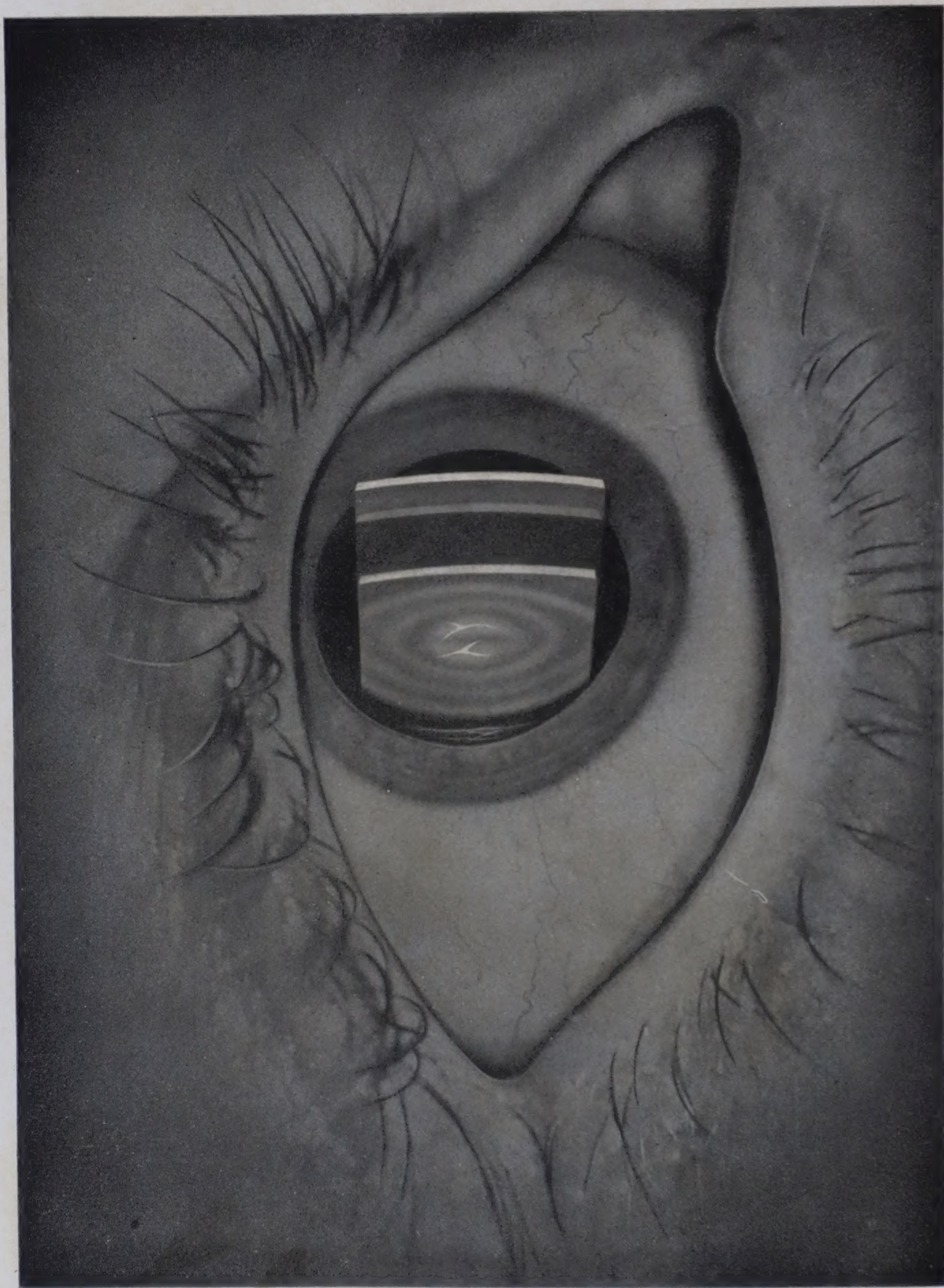
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BIOMICROSCOPY OF THE EYE

Slit Lamp Microscopy of the Living Eye

VOLUME I



FRONTISPIECE. Composite view, showing passage of slit beam through the transparent media of the eye. From left to right, corneal parallelepiped, dark interval representing depth of anterior chamber, lens block, and a small portion of the vitreous.

BIOMICROSCOPY OF THE EYE

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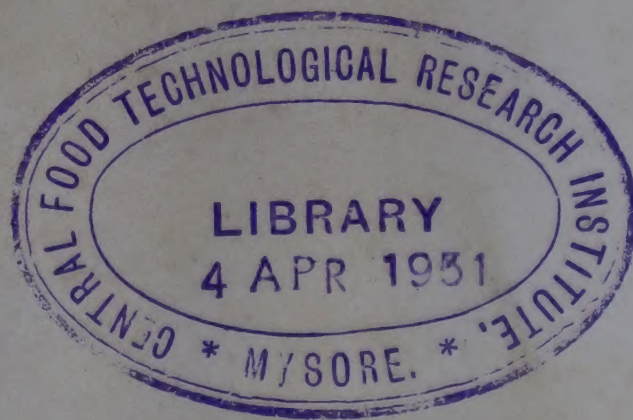
By

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WITH 1233 ILLUSTRATIONS INCLUDING 503 IN FULL COLOR

VOLUME I



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BIOMICROSCOPY OF THE EYE

VOLUME I

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Biomicroscopy of..

To
MY WIFE

PREFACE

DURING the last two decades the biomicroscope has ceased to be simply a research instrument. It now has an indispensable place in the armamentarium of the clinical ophthalmologist comparable with that of the ophthalmoscope. This importance has been recognized, not only by its established position in all postgraduate courses, but also by the fact that knowledge of its use is now required from all those taking the examinations of the American Board of Ophthalmology.

Before the development of biomicroscopy, understanding of the morphology of normal and pathologic changes in the transparent ocular media of the living eye depended chiefly on observation with ordinary oblique illumination or with the ophthalmoscope. I shall not dwell on the inadequacies of these methods, but I am sure that no one at this time will dispute the superiority of the combined use of the focal beam (as devised by Gullstrand) and the binocular microscope (biomicroscope) for examination of the living eye. With biomicroscopy not only can the normal living structure of the conjunctiva, cornea, lens, and vitreous be studied to a degree formerly impossible but finest alterations in them can be seen.

Daily observation of pathologic changes from inception to complete development is feasible with biomicroscopy, and this has afforded valuable insight in the diagnosis, prognosis, and treatment of many conditions. However, the accuracy of many interpretations of phenomena seen biomicroscopically must wait further critical analysis. Advances in biomicroscopy will depend not only on improvement in the instrument and in the technique of using it, but also on correlation of these improvements with new data obtained from physiochemical and histologic research.

From experience in teaching the technique of biomicroscopy to students for over fifteen years, I have found that there is a genuine need for a practical treatise on this subject. Such a textbook should describe the actual handling of the necessary apparatus and interpret the observations in the light of modern histopathologic knowledge. I realize the danger in overemphasizing one particular method of ophthalmologic examination, and that in doing so one must of necessity present only part of the whole. In these volumes, for the purposes of orientation and review I have given short definitions and condensed descriptions of various clinical entities occurring in the conjunctiva, eyelids, cornea, and anterior chambers; and have purposely avoided controversial matters of theoretic interest and some of the involved and highly technical explanations of physico-optic phenomena. In this reprint of the first volume Dr. Sugar has revised and enlarged his excellent chapter on Gonioscopy, and I have added two illustrations, of an aqueous vein and of pupillary membranes.

Volume II contains a similar discussion of the iris, vitreous, and lens, and a description of special methods of illumination and of methods for the examination of the deeper vitreous and fundus.

Lately there has been a great revival of interest in biomicroscopy of the deeper vitreous and fundus. Although the principles employed in the technique of observing these parts by means of optic section is not new, certain improvements have been developed. For example, the mirror used for narrowing the angle between illumination and observation has been supplanted by a prism (Goldmann), and Dr. Priestley and I have been experimenting with different types of prism for this use. Also the new forms of contact lens have been experimented with. Recently the lens (-50.00 diopters) originally suggested by Lemoine and Valois and modified by Hruby, which is placed before the eye in a frame, has obviated the more troublesome contact lens. The ability to obtain optical sections of the deeper vitreous and fundus may open up new fields in the diagnosis and understanding of conditions found in these parts.

One cannot examine the literature on the subject without becoming aware of the pathfinding and magnificent achievements of Al-

fred Vogt. His name is synonymous with almost every major advance made in biomicroscopy.

The appearance of the second volume of this work has been delayed by the war and its aftermath, and I have taken the opportunity to revise and bring up to date the material in it.

Since it is impossible to obtain adequate photographs, I have prepared drawings and colored illustrations of typical cases. In order to make this volume on biomicroscopy complete, frequent reference has been made to the outstanding works of Vogt, Koeppe, Koby, Graves, Meesmann, Goldmann, Harrison Butler, López-Lacarrère, and Duke-Elder. Specific citations are made in the text. I am indebted to the many colleagues and to the officials of the optical companies, whose permission to reproduce illustrations previously published is acknowledged more specifically in the text.

I wish to thank especially Mr. J. McGuinness Myers for the long and tedious hours spent in making the illustrations; and Dr. H. Saul Sugar for his valuable chapter on gonioscopy.

I also wish to express appreciation to Dr. Bruno Priestley, Dr. Lester Stein, Dr. Morton Biskind, and Miss Helen F. Roberts, and Miss Margaret G. Fiske for their assistance in preparing the manuscript.

The color plates of these two volumes have been made by the Offset Printing Plate Company of New York, Inc., and the black and white illustrations by the Lotz Photoengraving Company of Philadelphia, to both of whom go my sincere thanks.

New York, N. Y.

M. L. BERLINER

CONTENTS

VOLUME I

PREFACE	vii
I. DEVELOPMENT OF BIOMICROSCOPY	1
II. THE BIOMICROSCOPE	14
The Illuminating System — Optical Characteristics of the Luminous Beam — The Stereoscopic Microscope — Czapski Microscope (Zeiss Model) — Comberg Slit Lamp — Universal Slit Lamp — Simplified Universal Slit Lamp — Poser Biomicroscope — Other Biomicroscopes — Hand Slit Lamps	
III. TECHNIQUE OF BIOMICROSCOPY	64
Preliminary Survey of Eyeball — Illumination of Room — Preparation and Management of the Patient — Adjustment of the Instrument — Stereopsis — Methods of Illumination — Projection of Shadows — Methods of Localization — Procedure for Efficient Biomicroscopy of the Eye	
IV. THE NORMAL CONJUNCTIVA	124
Histology — Blood Supply — Circulation of the Conjunctival Vessels — Technique of Biomicroscopic Examination — Biomicroscopy of the Normal Conjunctiva — Vital Staining of the Conjunctiva	
V. NONINFLAMMATORY LESIONS OF THE CONJUNCTIVA	154
Developmental Anomalies of the Conjunctiva — Degenerative Changes of the Conjunctiva — Endogenous Pigmentation of the Conjunctiva — Exogenous Pigmentation of the Conjunctiva	

XVII. GONIOSCOPY	<i>H. Saul Sugar</i>	598
Gonioscopic Technique and Apparatus — Goniometry — Clinical Value of Gonioscopy and Its Limitations — Normal Anatomy of the Angle of the Anterior Chamber — Congenital Alterations in the Anatomy of the Chamber Angle — Pathologic Alterations in the Chamber Angle		
BIBLIOGRAPHY FOR VOLUME I		671
INDEX TO VOLUME I		693

VOLUME II

XVIII. THE IRIS		727
Morphology — Methods of Examination — Biomicroscopic Appearances of the Normal Iris — Iris Coloration — Size and Mobility of the Pupil — The Senile Iris		
XIX. CONGENITAL ANOMALIES OF THE IRIS		779
Anomalies of the Pupillary Pigment Border (Seam) — Defects Principally Involving the Stroma — Anomalies of Pigmentation in the Iris		
XX. INFLAMMATION OF THE IRIS		808
Hyperemia — Exudation in the Anterior Chamber — Pigmentary Changes — Iris (Stromal) Atrophy (Postinflammatory)		
XXI. SPECIFIC TYPES OF IRITIS		869
Herpetic Iritis — Gonococcal Iritis and Iridocyclitis — Syphilitic Iritis and Iridocyclitis — Tuberculous Iritis and Iridocyclitis — Leprosy of the Iris — Heterochromic Iridocyclitis — Sympathetic Ophthalmitis — Essential Progressive Atrophy of the Iris — Polycythemia Vera (Vaquez-Osler Disease) — Lymphoma		

XXII.	TRAUMATIC LESIONS, CYSTS, AND TUMORS OF THE IRIS	912
	Traumatic (Mechanical) Lesions — Cysts — Tumors — Leiomyoma — Angiomas — Malignant Melanoma of the Iris — Leukosarcomas — Metastatic Carcinoma of the Iris	
XXIII.	THE NORMAL LENS; BIOMICROSCOPIC APPEAR- ANCE	948
	The Lens — Technique of Biomicroscopy of the Lens — Relationship of the Morphology of the Lens to its Function of Accommodation — Modifications Associated with Aging — Physio- logic Remains of the Tunica Vasculosa Lentis	
XXIV.	DEVELOPMENTAL LENS CHANGES	1032
	Congenital Anomalies of the Lens — Gross Changes in Size, Shape, or Position of the Lens — Developmental Cataracts	
XXV.	PRESENILE AND SENILE CATARACT	1091
	Coronary Cataract — Senile Cataract — Corti- cal Cataract — Mature Cataract — Hyper- mature Cataract — Cupuliform Cataract — Nuclear Cataract	
XXVI.	COMPLICATED CATARACT	1159
	Biomicroscopic Appearance of Cataracta Com- plicata — Cataracts Associated with Endocrine Disorders — Cataract of Myotonia Dystrophia — Cataract Associated with Mongolian Idiocy — Cataract Associated with Certain Skin Diseases — Toxic Cataracts — Cataract Associ- ated with Glaucoma — Deformations of the Lens by Local Pressure from Tumors of the Ciliary Body — Spontaneous Dislocation of the Lens — Certain Pathologic Alterations of the Lens Cap- sule	
XXVII.	INJURIES TO THE LENS	1235
	Contusion Cataract — Traumatic Cataract As- sociated with Rupture of the Lens Capsule —	

	Lens Changes Following Penetrating Foreign Bodies	
XXVIII.	RADIATION CATARACT	1291
	Heat ("Fire") Cataract; Infra-red Cataract — Roentgen (X-ray) and Radium Cataract — Biomicroscopic Appearance of X-ray and Radium Cataract — Electric Cataract (Cataracts from Lightning and Electric Shock)	
XXIX.	SECONDARY CATARACT	1310
	Secondary Membrane — After-cataract — Cataracta Secundaria	
XXX.	THE ZONULE	1327
	General Considerations — The Zonule in Certain Hereditary and Congenital Anomalies — Pathologic Alterations of the Zonule Following Inflammation — Traumatic Alterations of the Zonule	
XXXI.	THE VITREOUS	1362
	General Considerations — Technique of Biomicroscopic Examination of the Vitreous — Biomicroscopic Appearance of the Normal Vitreous — Special Methods Devised for the Examination of the Vitreous — Congenital Anomalies	
XXXII.	BIOMICROSCOPY OF VITREOUS OPACITIES	1401
	General Considerations — Senile and Myopic Alterations — Synchysis Scintillans and Asteroid Hyalosis — Changes in Inflammation — Tumors Affecting the Vitreous — Traumatic Injuries to the Vitreous — Intra-ocular Parasites	
	BIBLIOGRAPHY FOR VOLUME II	1467
	INDEX TO VOLUME II	1489

BIOMICROSCOPY OF THE EYE

Slit Lamp Microscopy of the Living Eye

Chapter One

DEVELOPMENT OF BIOMICROSCOPY

ON August 3, 1911, Alvar Gullstrand presented his first rudimentary model of the slit lamp before the "Versammlung der deutschen ophthalmologischen Gesellschaft" in Heidelberg, and explained its optics and applications. His discovery of a method of producing a truly focused beam of light marked the climax of a period, lasting 150 years, in which ophthalmologists had struggled to find a satisfactory clinical method of illumination for examining the anterior parts of the living eye.

During the Middle Ages, owing to the poor light available from candles or oil lamps, it was necessary to use daylight for examination or surgery of the eye. At the beginning of the nineteenth century, Himly (1772-1837) reported that oblique or condensed focal illumination of the eye made more accurate observation possible. In 1806 Himly and William MacKenzie of Glasgow also emphasized the value of this form of lighting. Later, in 1823, Purkinje employed a lens to magnify the details of the iris under strong oblique illumination.

The full significance of oblique illumination of the eye was not stressed until almost fifty years later, when Helmholtz described its theoretical use in an essay, "On Accommodation of the Eye" (1854): "The subject sits on a bench, his chin firmly resting on a support. The observer places a lamp laterally from the observed eye, in such a way so as to illuminate the cornea and the iris. The collarette projects a shadow which is easy to follow."

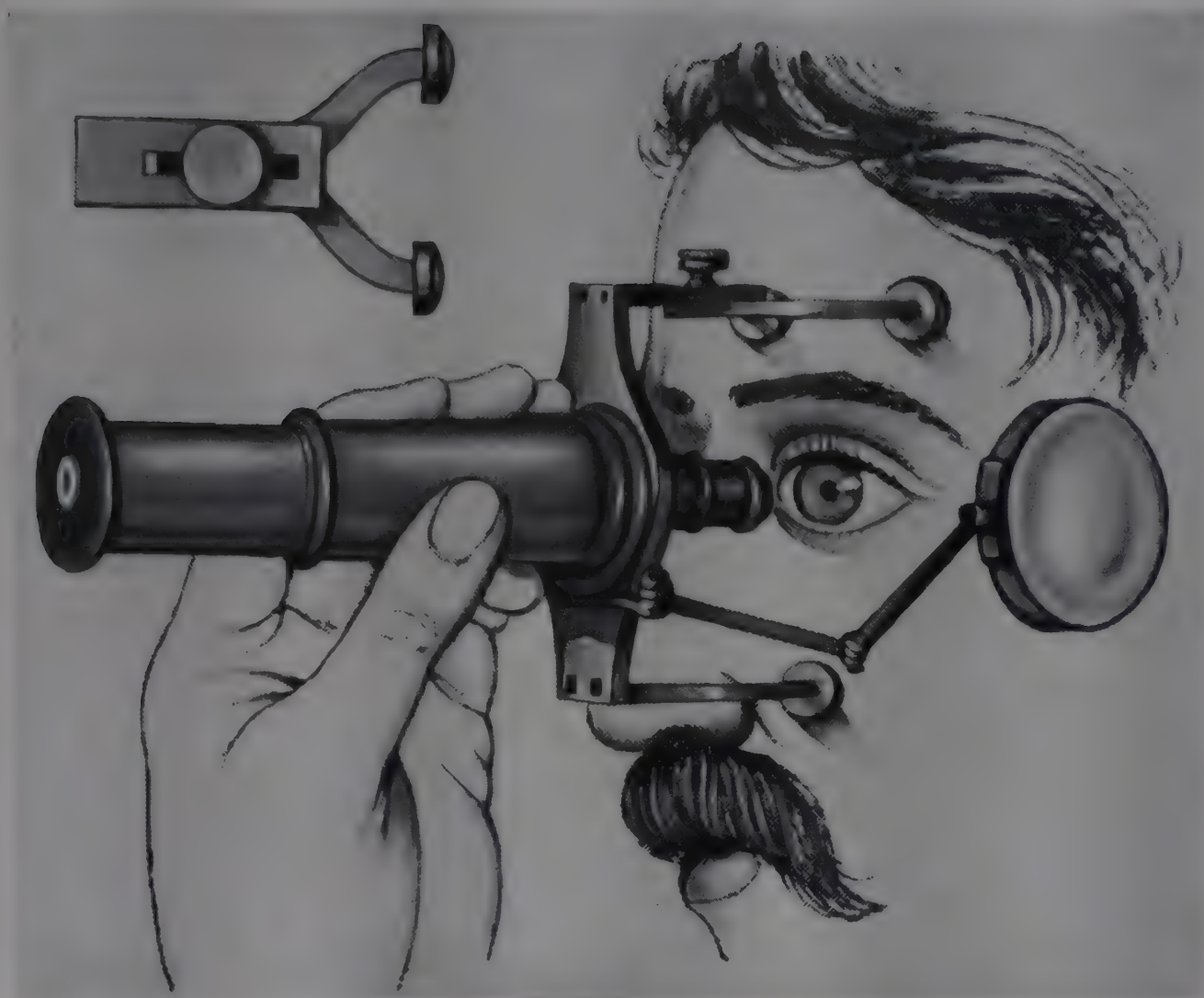
Helmholtz was interested only in the processes of accommodation as they affected form, size, location of light, images and shadows.

He did not use oblique illumination for examining the ocular tissues per se.

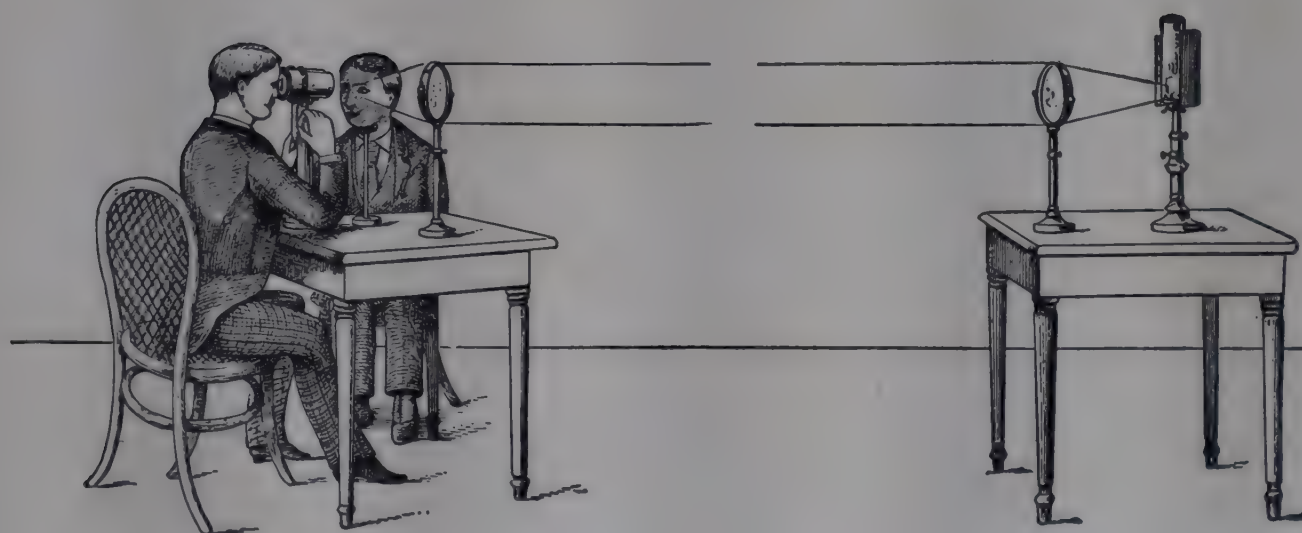
Oblique illumination was advocated for clinical use by von Graefe¹²⁵ in 1854, in an article on cataracts, in which he stated that "the appearance of a yellowish nucleus in the lens is observed by means of an artificial light falling obliquely upon the eye; a method, which I believe, is of outstanding value for the examination of the capsule and the lens." Liebreich, in the same issue of the *Archiv für Ophthalmologie*, presented a comprehensive discussion of the method and suggested its adoption by all ophthalmologists. He was the first to employ a microscope and to recommend the use of different portions of a beam (prefocal and postfocal) obtained by oblique illumination. Liebreich's technique consisted in the combined use of a magnifying loupe or simple microscope for observation with a condensing lens to focus the light. With the aid of the Schick microscope he obtained magnifications up to 90 \times . Liebreich emphasized the importance of the focal part of the beam for the observation of fine details and cautioned that "the angle between the axis of the beam and the axis of observation should be wider when looking at the equatorial part of the lens, than when examining the posterior pole of the lens or vitreous. In both cases very small opacities will be recognized in their true color and exact position."

Other European workers advocated the combined use of a magnifying system and oblique (condensed) illumination (Fig. 1). As early as 1863, de Wecker devised an apparatus called the ophthalmomicroscope, in which a Hartnack microscope was fastened to a three-legged support, which rested against the periorbital region. This microscope was used in conjunction with a condensing lens in order to obtain concentrated illumination. Gayet perfected de Wecker's early model by adding two lenses, one of which acted as a condenser and the other as a projection lens.

In 1891, H. Aubert presented a binocular corneal microscope at the Ophthalmological Congress in Heidelberg. This microscope, later modified by Czapski (1897), is the type still used, but then it was combined with the Aubert low-voltage electric lamp, which afforded



A



B

FIG. 1. A. De Wecker ophthalmomicroscope. B. Laqueur's method for examination of the eye by lateral illumination. Magnification of $\times 10$ was obtained with Westien's loupe.

oblique illumination in a more or less diffuse form. The lamp was arranged so that it could be moved along a rail (Lucanus) (Fig. 2).



FIG. 2. The corneal microscope with Lucanus curved illuminating rail. (Courtesy of Carl Zeiss, Inc.)

Although other ophthalmologists constructed similar devices, they were soon compelled to recognize that beyond a certain point, it was useless to employ greater magnification and brighter sources of illumination, since even these aids could not reveal new details. The fault lay with the method of illumination; for as Gullstrand later demonstrated, it was impossible to obtain a truly focused beam by using oblique illumination as provided by a simple condensing lens (page 6). In "System of Diseases of the Eye" (1897), edited by Norris and Oliver, Laqueur¹⁸⁹ discussed the value of the corneal microscope (Aubert-Czapski) as follows: "It was quite natural to think of applying the microscope to the examination of the living eye, and great hopes were built upon it. They have not, however, been realized, and they can never be realized, because of insurmountable difficulties. The short focal distance—less than one

centimetre, even with low powers — and the unavoidable slight movements of the eye cause mechanical irritation of the eyelids and injuries of the cornea that may be serious. Furthermore, the field is so much narrowed that it becomes very difficult to find any particular spot. The greatest difficulty, however, is that owing to movements of the eye, any part can be rarely observed for half a minute before it disappears out of the field or out of the area of illumination. This difficulty is less pronounced in Aubert's microscope, but even with this instrument it is not entirely avoided. The use of the corneal microscope is illusory. There is no new fact that owes its discovery to the microscope in its application to the living eye. Whatever can be seen by its means can be seen more easily and surely with the binocular corneal loupe. For this reason it is rarely used, and it is doubtful whether it will ever be useful in examining the living eye."

Because of such opinions and because the method is simple, most ophthalmologists employed the "loupe and lens" for observing the anterior ocular structures (Fig. 3). This consists of a biconvex condensing lens of from $+12$ to $+15$, diopters, which forms an oblique beam by concentrating light from a near-by lamp. The parts so illuminated are then viewed with a monocular ophthalmic loupe, varying from $6\times$ to $10\times$. Stereoscopic effects can be obtained by the use of various types of binocular loupes, in which prisms are incorporated. These binocular loupes, though they provide lower magnifications, have the advantage of having an attached head band (Berger loupe) or of being worn as spectacles, thus leaving one hand free.

A technique for the production of a focal beam for illumination of the retina was described by H. Wolff. He constructed an ophthalmoscope with a carbon filament bulb. The light from this source, which passed through a condensing lens, was reflected into the eye by means of an oblique mirror and cast an image of the filament on the retina. This was the first ophthalmic instrument in which a truly focused beam, as it is now understood, was employed for the examination of ocular tissue.

No further efforts to adapt this form of focal illumination to the study of the ocular media were made until those of Gullstrand (1905-1911).¹⁴⁰ While he was conducting his investigations the



FIG. 3. Method of ordinary oblique illumination with a condensing lens.

Nernst lamp became available. The filament of this lamp was composed of a tightly coiled tungsten spiral, coated with a compound of metallic oxides, which when electrified, became incandescent; it was rod-shaped and, therefore, peculiarly suitable for use with a slit diaphragm. Gullstrand recognized its value and employed it widely in his researches. These led ultimately to the development of his method of projecting the image of the "Nernst" rod in a slit opening, thereby producing a rectangular focal beam. Gullstrand states in "Einführung in die Methoden der Dioptrik": "With the focal light system currently used in ophthalmic practice the necessary

brilliancy is obtained with a convex lens which forms a small image of the light source at a distance from the object being illuminated. Owing to the aberration of common lenses and to the larger size of common light sources, when the optic image of the light source is put directly on the object, or very near it in order to improve the brilliancy, one sees a design which comes either from the light source or from the caustic surface* of the object which obscures details and interferes with observation. Only when we obtain a really focused light by means of the *Nernst-spaltlampe* and aplanatic lens are we able to overcome these drawbacks."

Gullstrand showed the great advantage of projecting the rays of light emanating from an *image* of the light source rather than those emitted by the luminous body itself. By means of this method it became possible to obtain an easily controllable beam of strongly focused light without the disadvantage of ordinary oblique illumination. This contribution made biomicroscopy of the eye a possibility.

Gullstrand's original instrument consisted chiefly of a tube at one end of which a Nernst bulb was inserted; at the other end there was a diaphragm with a slit opening in which a real image of the glowing filament was centered by means of a condensing lens system (Fig. 4). He was thus able to obtain a small beam of light of high intensity which was easily handled. This system combined with an aplanatic lens produced a maximum degree of focal illumination. Gullstrand originally designed the apparatus to make ophthalmometric examinations of the corneal curvatures and only incidentally mentioned its value for general diagnostic purposes. He also said that it could be modified and used for transillumination of the vitreous.²²⁹ Apparently at this time Gullstrand was searching for a small but brilliant source of light for focal illumination which could be used for various optical purposes in connection with his studies of the eye; he did not realize until later the full value of the practical application of his method of focal illumination to the study of pathologic changes of the eye.

* By caustic surface is meant the envelope of rays emanating from a focus and reflected or refracted by a curved surface. (The envelope is called a caustic surface.)

In 1916, Henker developed the prototype of the modern biomicroscope by mounting the illuminating system of Gullstrand on a horizontal rigid swinging arm in conjunction with the Czapski corneal microscope (Fig. 5).



FIG. 4. Gullstrand's original model of the Nernst slit lamp illuminating unit. Constructed in 1908 and originally used for measuring the curvature of the posterior corneal surface. In 1909 it was employed in the Upsala Clinic as a source of focal illumination for examining the transparent ocular media. A photograph of this instrument has never previously been published. (Courtesy of Professor J. W. Nordensen.)

In 1920, Vogt³²¹ adapted Kohler's system of illumination (1893) to the biomicroscope, using a coiled tungsten filament nitra bulb. By this method the image of the filament light source is focused on the surface of the illuminating lens instead of in the slit, thus obviating the projection of the spiral filament directly on the eye. Later Koeppe, Koby, López-Lacarrère, Comberg, Arruga, Poser, and others added further refinements to the biomicroscope.

Among English workers, Basil Graves is outstanding for his contributions to the terminology and interpretation of biomicroscopic phenomena. Butler⁴² in 1927 wrote the first English textbook on

biomicroscopy, entitled "An Illustrated Guide to the Slit Lamp."

The discussion of the "Phenomena of Reflection of Light by the Ocular Media" in the second edition of Koby's "Slit Lamp Mi-

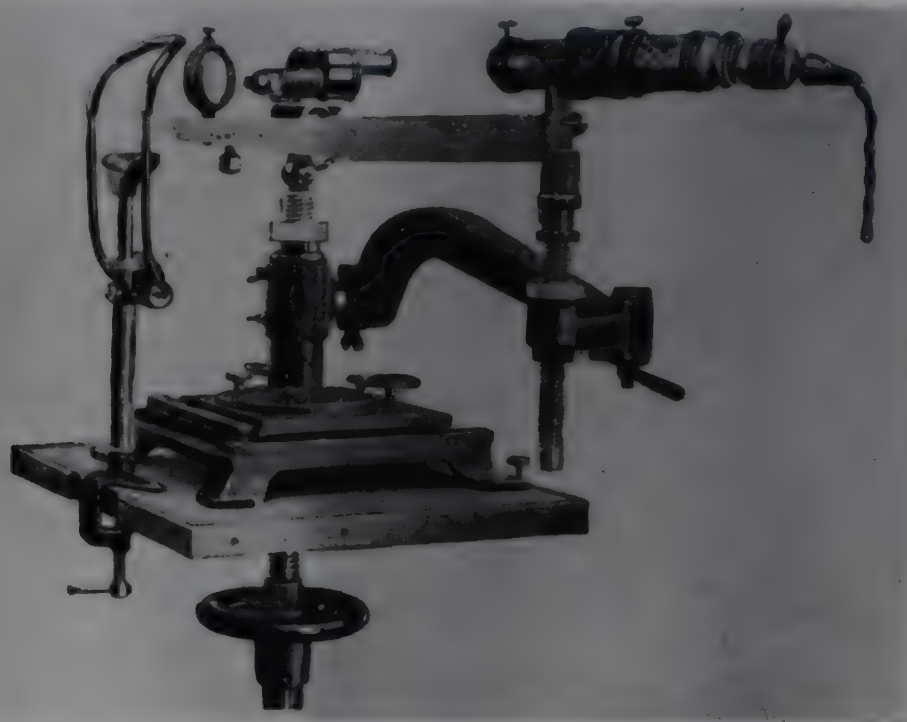


FIG. 5. Henker model with articulated arm for mounting Gullstrand's illumination apparatus.
(Courtesy of Carl Zeiss, Inc.)

croscopy of the Living Eye"¹⁷⁵ is of incalculable value in elucidating many obscure and difficult problems in biomicroscopy.

Essentially, the principle of biomicroscopy depends on full utilization of focal illumination, that is, light projected by a condensing lens or system of lenses (in contradistinction to diffuse illumination). At the focal point of such a system, light becomes intensely concentrated. However, as Gullstrand so clearly showed (page 6) the oblique beam, as ordinarily obtained (by means of a simple condensing lens, i.e., oblique illumination), is inadequate and even obscures details. But with the beam which he devised and which was modified by Vogt, it is possible to examine the successive layers of the transparent ocular media illuminated by light in exact focus (Fig. 6). As the beam of focused light passes through the transparent media of the eye, only a small section of tissues is illuminated, corresponding to the size and shape of the beam. Consequently, there is a marked contrast between the illuminated area and the surrounding area in shadow. It may be compared to the passage of a

beam from a searchlight through the night sky. Any object in the path of such a beam is immediately illuminated and becomes visible. Another example of this phenomenon is noted when a sunbeam



FIG. 6. The successive zones of the transparent ocular media as illuminated by direct focal illumination.

enters a darkened room through a small crack and dust motes in its path may actually be seen suspended in the air. This phenomenon bears the name of Tyndall, who was the first to call attention to it. In one of his important works, Tyndall states: "For fifteen years it has been my habit to make use of floating dust to reveal the paths of luminous beams through the air, until 1868 I did not intentionally reverse the process and employ a luminous beam to reveal and examine the dust. The experiment proved that no sensible amount of light was scattered by the molecules of the eye; that the scattered light always arose from suspended particles."

The principle of this phenomenon is utilized in the ultramicroscope which renders suspended particles visible by means of an intense beam of light directed across the field of the objective at right angles to the axis of the microscope. Each particle scatters some of this light, and the scattered light, which enters the objective from any one particle, is focused as a diffraction disk in the image plane of the microscope ocular. These otherwise invisible particles are thus rendered visible to the eye by means of increased intensities of light

and the apparent increase in size obtained by the formation of diffraction disks.

A similar effect is produced by projecting an intense pencil of light into the eye with the biomicroscope. Transparent tissues, like the cornea, lens and vitreous, are actually gels and hence will show the Tyndall phenomenon in varying degrees. The phenomenon becomes more apparent in pathologic conditions (Fig. 7). By utilizing these diffraction phenomena, visualization of details is enhanced optically, and this tends, to a degree, to obviate the necessity of having high resolving powers in the microscope.

In contrast to ordinary histologic microscopy, in which high magnifications are necessary in order to observe cellular details, the biomicroscope permits, for instance, visualization of floating cells in the aqueous fluid with a magnification as low as $22\times$. In addition to observation with the focal beam per se the biomicroscope makes possible the study of the transparent media by means of scattered light, internally reflected light (retro-illumination), surface reflected light (specular reflection), and combinations of these.

The intrinsic optic properties of the ocular media make these methods of illumination possible. The study of zones of specular reflection (Vogt), that is, the reflections of the mirror-like surfaces of the cornea and lens, permits the examiner to observe these surfaces with results previously unobtainable by other methods of illumination.

From time to time, many names and definitions have been proposed and rejected for this branch of ophthalmologic examination. Originally, the instrument was known as the Gullstrand slit lamp microscope, but this was soon contracted to "slit lamp." It was also called the "corneal microscope." This term is incorrect because the use of the Czapski microscope is not limited to study of the cornea. Similarly, the term "slit lamp" is inaccurate since the slit is only one of the types of diaphragmatic openings available. In 1925, Mawas²¹⁶ recommended the name "biomicroscopy," stating that "biomicroscopy is short, accurate and substitutes advantageously for the phrase: examination of the living eye by means of



FIG. 7. Tyndall phenomenon in a case of turbid aqueous in cyclitis. C, Cornea;
A. C., anterior chamber; L, lens. (Cylindrical beam.)

corneal microscope and slit lamp." Jackson¹⁵⁸ stated that "a single word is needed to designate this method of examination; to call it the 'slit lamp' examination, using two words instead of one descends to a barbarism in language. It is suggestive of a temporary phase of development. The term 'biomicroscopy' is broad enough to include any future change of instrument, method, or details. It focuses attention on the essential character of the examination of living tissue with the microscope." Therefore, throughout this work the term biomicroscopy will be used. In short, for the purpose of definition biomicroscopy in a restrictive sense may be considered an intravital, histologic method for the study of the ocular tissues.

Chapter Two

THE BIOMICROSCOPE

THE apparatus originally devised by Gullstrand, with which it was necessary to hold an illuminating lens with one hand and a single or binocular loupe with the other, was awkward to handle. Credit must be given to Henker,¹⁴⁹ who conceived the idea of combining the Gullstrand illuminating system with the Czapski microscope. He mounted the various parts of the illumination system on a horizontal arm articulated to a table; the microscope rested on this table and a chin support was attached to it (Figs. 5, 8).

The description of the early apparatus may be discussed under two headings: (1) the illuminating system and (2) the stereoscopic microscope. The illuminating system consists of: (a) the source of illumination; (b) the condensing lens; (c) the diaphragm; (d) Koeppe's tube; (e) the illuminating lens.

THE ILLUMINATING SYSTEM

THE SOURCE OF ILLUMINATION

The original source of illumination was the Nernst lamp. Although the Nernst lamp gave a bright and homogeneous light, it was unsatisfactory in use because of its fragility and short life. The bulb was enclosed in an adjustable housing, which had a sliding sleeve to focus the light and screws for centering it. The Nernst rod, a tungsten spiral coated with magnesium, cerium and thorium oxides, was 2 cm. long and from 0.5 to 1 cm. wide. In order to illuminate the rod, it was necessary to raise the temperature of the filament to 600° C. which took considerable time. This was accomplished by the use of a platinum resistance coil.

Today, the nitrogen-filled "nitra" lamp is used exclusively (Figs.

10, 11). It is easily obtainable, more durable, and less expensive. The nitra lamp consists of a coiled tungsten filament in a bulb filled with nitrogen. It operates on ordinary house current, which

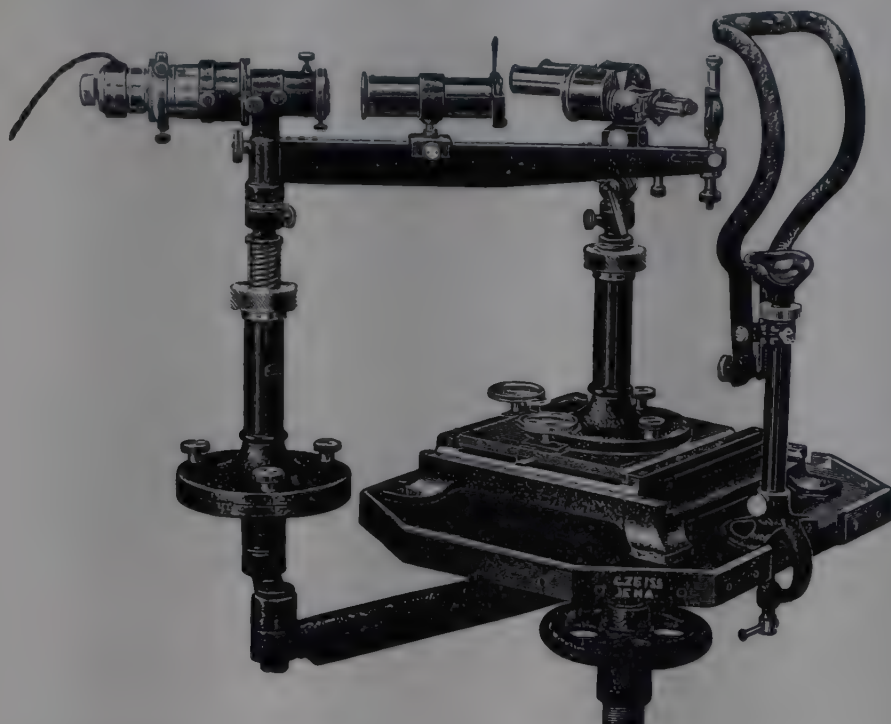


FIG. 8. Zeiss biomicroscope with mechanical stage. (Courtesy of Carl Zeiss, Inc.)

is reduced by a rheostat or transformer (for alternating current) to from 8 to 10 volts. It is possible to obtain stronger illumination by overloading the lamp to from 10 to 12 volts, but this shortens the life of the bulb. However, the higher voltage is an advantage in studying the vitreous. Other types of illuminants are: the arc

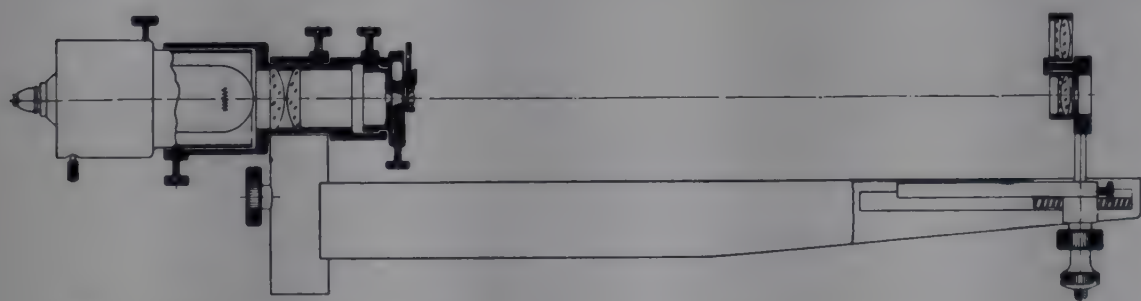


FIG. 9. Diagram of longitudinal section of the Zeiss illuminating system. (Courtesy of Carl Zeiss, Inc.)

lamp, the mercury vapor lamp for monochromatic illumination, and the quartz ultraviolet lamp for fluorescent microscopy.

The arc lamp produces a beam of the greatest luminous intensity. When using the narrow slit and the nitra lamp the luminous intensity of the beam is diminished and, therefore, the greater luminos-

ity obtained from an arc is especially valuable for observation of the deeper tissues. On the other hand, the arc lamp has several drawbacks. The carbons, despite automatic feeding, require constant

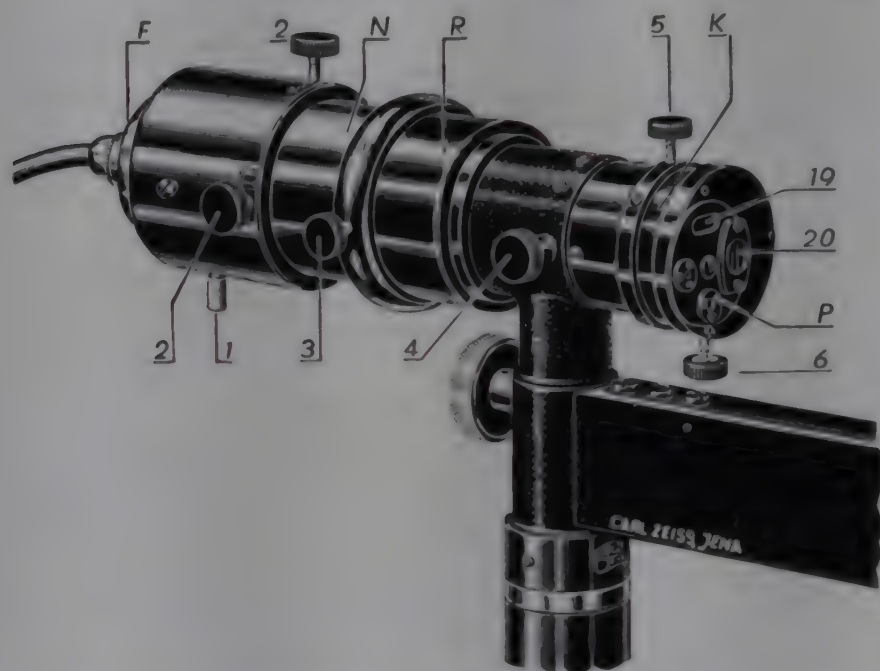


FIG. 10. The nitra lamp illuminating unit. *N*, Removable nitra lamp housing; 2, adjusting screws; 3, clamping screw. *R*, Slit lamp tube, with clamping screw (4). *K*, Revolving slit lamp head, with clamp screw (5) and slit width adjusting screw (6). *P*, Revolving diaphragm disk, with open aperture (19) and diaphragm with Meesmann lens (20). (Courtesy of Carl Zeiss, Inc.)

manipulation to assure their proper position. Moreover, in addition to being cumbersome, the arc radiates considerable heat. Several housings have been devised which are readily interchangeable with those of the usual nitra lamp (Fig. 12). Birkhäuser³⁰ developed a

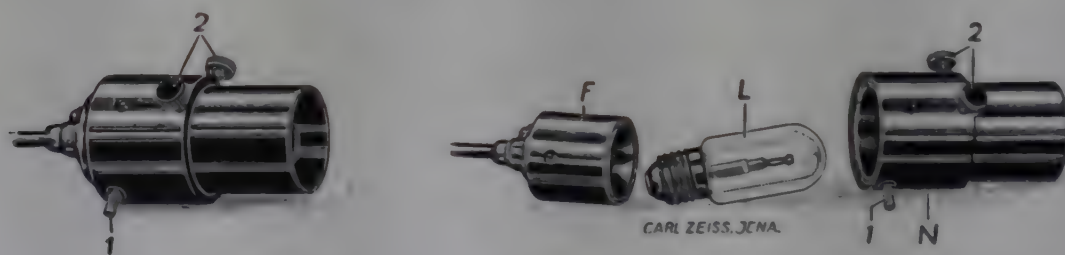


FIG. 11. Nitra lamp and housing, assembled and dismantled. *F*, Lamp holder; *L*, nitra lamp; *N*, lamp tube with centering screws (2) for adjusting the nitra lamp and clamping screw (1) for clamping the lens holder (*F*) in the lamp tube. (Courtesy of Carl Zeiss, Inc.)

carbon arc lamp* which differed from the Vogt (Zeiss) arc unit in that it was provided with rollers to facilitate sagittal focusing of the beams.

Duke-Elder⁶² designed a mercury vapor lamp which could be adapted to either diagnostic or therapeutic purposes (Fig. 15). The

* Manufactured by A. Streit, Berne.

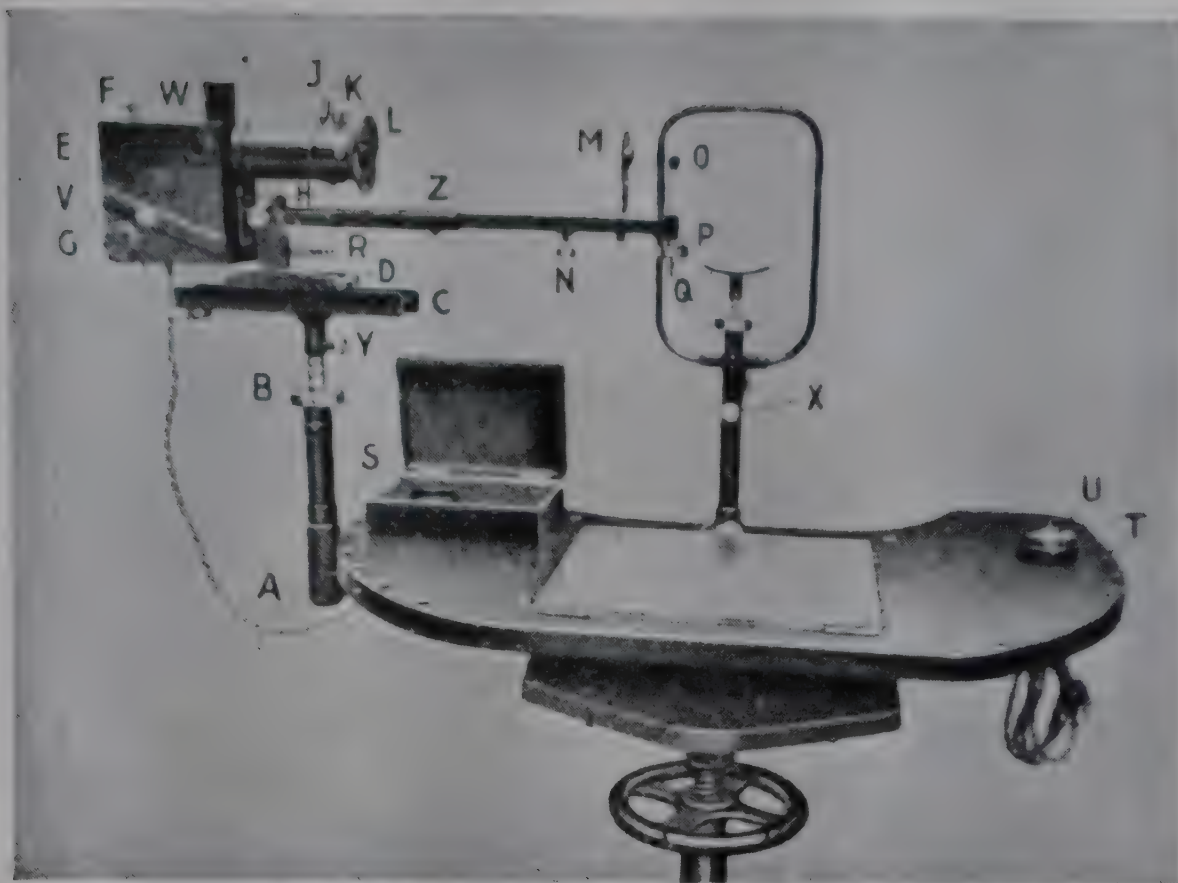


FIG. 12. Birkhäuser arc lamp. *T*, indicates the incidence of the luminous beam. *U*, light switch. The lamp slides sagittally on slide *C*. The arm (*Z*) is subject to movement which cannot be regulated. The lens (*M*) and the mirror (*O*) are of special design; likewise, the chin-rest and head-rest. The axis of rotation of the long arm of the lamp will be found under the chin-rest.



FIG. 13. Vogt arc lamp. (Courtesy of Carl Zeiss, Inc.)

spectrum of the mercury vapor lamp at the upper limit ends at 5790 angstrom units. This eliminates the infra-red rays and their resultant heat, which is such a disadvantage with the ordinary arc.

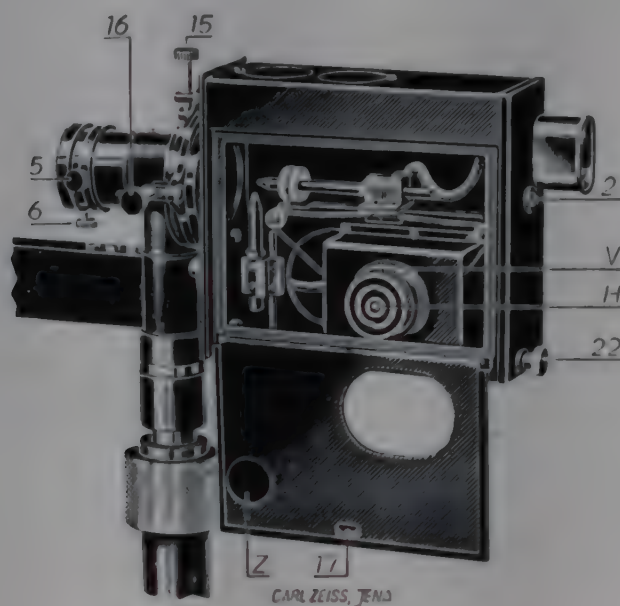


FIG. 14. Interior view of the Vogt arc lamp. The arc lamp opened, ready for inserting the carbons. V, Rear set knob for displacing both carbons together. H, Front set knob moving the horizontal carbon alone. 21, Aperture through which the horizontal carbon is inserted. 22, Adjusting head for setting the crater at the correct distance from the condenser, thereby focusing the crater image. (Courtesy of Carl Zeiss, Inc.)

For diagnostic purposes abiotically active light (under 3700 Å) must be filtered off (Fig. 16). Duke-Elder employed a Crookes filter, combined with a quartz ultraviolet lamp and quartz condensing lenses, to obtain a band of light which produced fluorescence in the eye. This light is projected into the eye in the same way as the ordinary nitra beam, but its use, also advocated by Thiel, is still mainly of academic interest. The fluorescent effect can be enhanced by giving the patient 2 gm. of sodium fluorescein by mouth thirty minutes before the examination. Not only the skin of the eyelids but also the normal cornea and lens fluoresce markedly, giving off a pale yellow-green or grayish lavender glow. Pathologic features show no fluorescence and appear black or blue in contrast to the vivid background. The lens fluoresces to a striking degree and since the absorptive properties of the nuclei vary from those of the cortex, these layers stand out by contrast. In cataract surgery, lenticular debris can easily be identified in the anterior chamber by the use of fluorescent ultraviolet light.

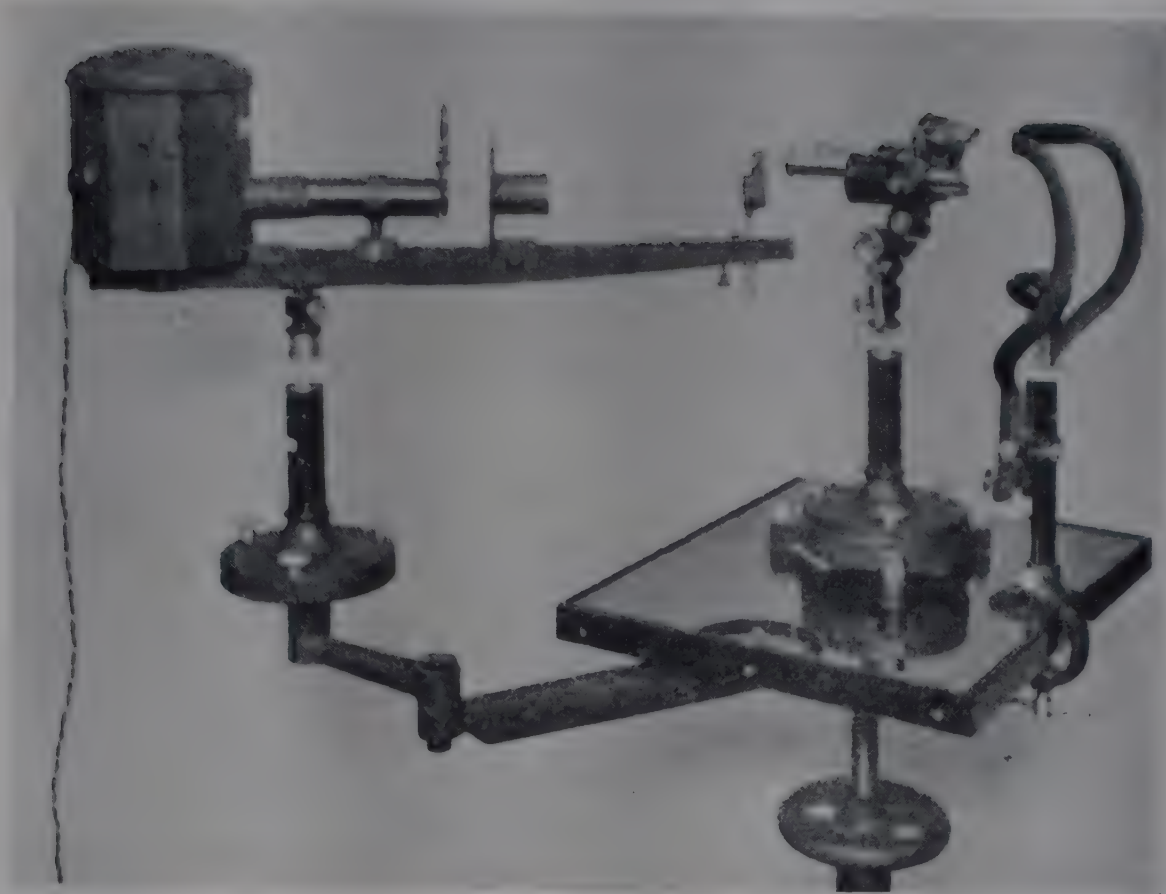


FIG. 15. Mercury vapor apparatus of Duke-Elder for obtaining ultraviolet illumination. (Duke-Elder, W. S. Recent Advances in Ophthalmology. Courtesy of J. & A. Churchill, Ltd.)

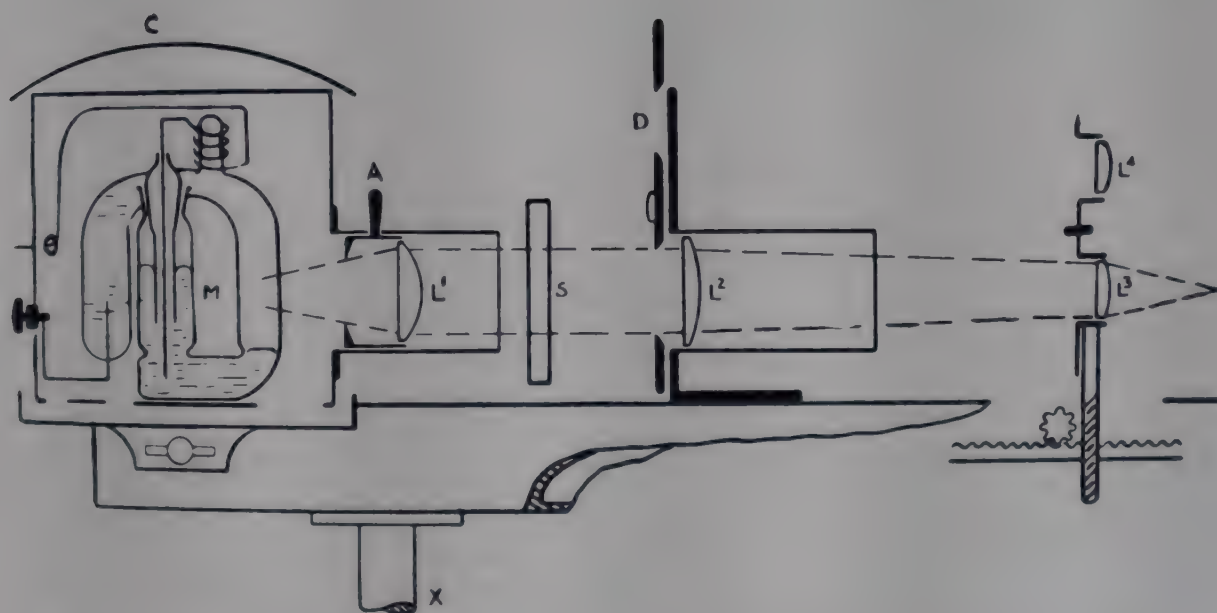


FIG. 16. Diagram showing construction and optics of the Duke-Elder mercury vapor lamp. (Duke-Elder, W. S. Recent Advances in Ophthalmology. Courtesy of J. & A. Churchill, Ltd.)

THE CONDENSING SYSTEM

The condensing system consists of two plano-convex lenses totaling 22 diopters. These are placed so that the curved surfaces are in apposition. The front focus of the condensing system falls on the illuminating lens.

In Gullstrand's method of illumination, the first condensing lens is placed with its principal focus at the lamp filament, while the second focuses parallel rays from the first on the slit diaphragm. In the Kohler-Vogt method,* the lamp and condensing system are brought closer together so that the image of the filament is sharply focused on the illuminating lens. The adjustable sleeve on the housing of the light source in the instrument permits easy interchange of the two methods of illumination by simply altering the distance of the illuminant from the condensing lenses. In Gullstrand's method the beam in its focal part is not as sharp or as bright or as large as in Vogt's method.

THE DIAPHRAGM

The collected light coming from the condensing lenses passes through one of the openings in a revolving circular disk (Fig. 10, 19 and 20). The rectangular opening, which is usually employed, is known as the slit aperture. By rotating the disk, stenopeic openings of different sizes may be obtained. The slit aperture is 10 mm. in height and when completely open measures 2 mm. in width. The width may be narrowed by a regulating screw to 0.5 mm., in which case the emergent beam measures 20 μ in its focal portion. In addition to the slit aperture, there are two stenopeic openings in the rotating disk, the respective sizes of which are 2 mm. and 1 mm. These produce conically shaped beams, the focal part of which Vogt considers to be cylindrical. López-Lacarrère¹⁹⁹ designed a modification in which a double slit diaphragm is employed, permitting the simultaneous use of a broad and a narrow beam (Figs. 17, 18). The broader opening has the form of an inverted L. The shape and recip-

* Devised in 1920 after a suggestion of Kohler, in 1893.

rocal position of the two openings can be changed by means of two screws as shown in Figure 17, thus offering the advantage of the wide and narrow beam at the same time.

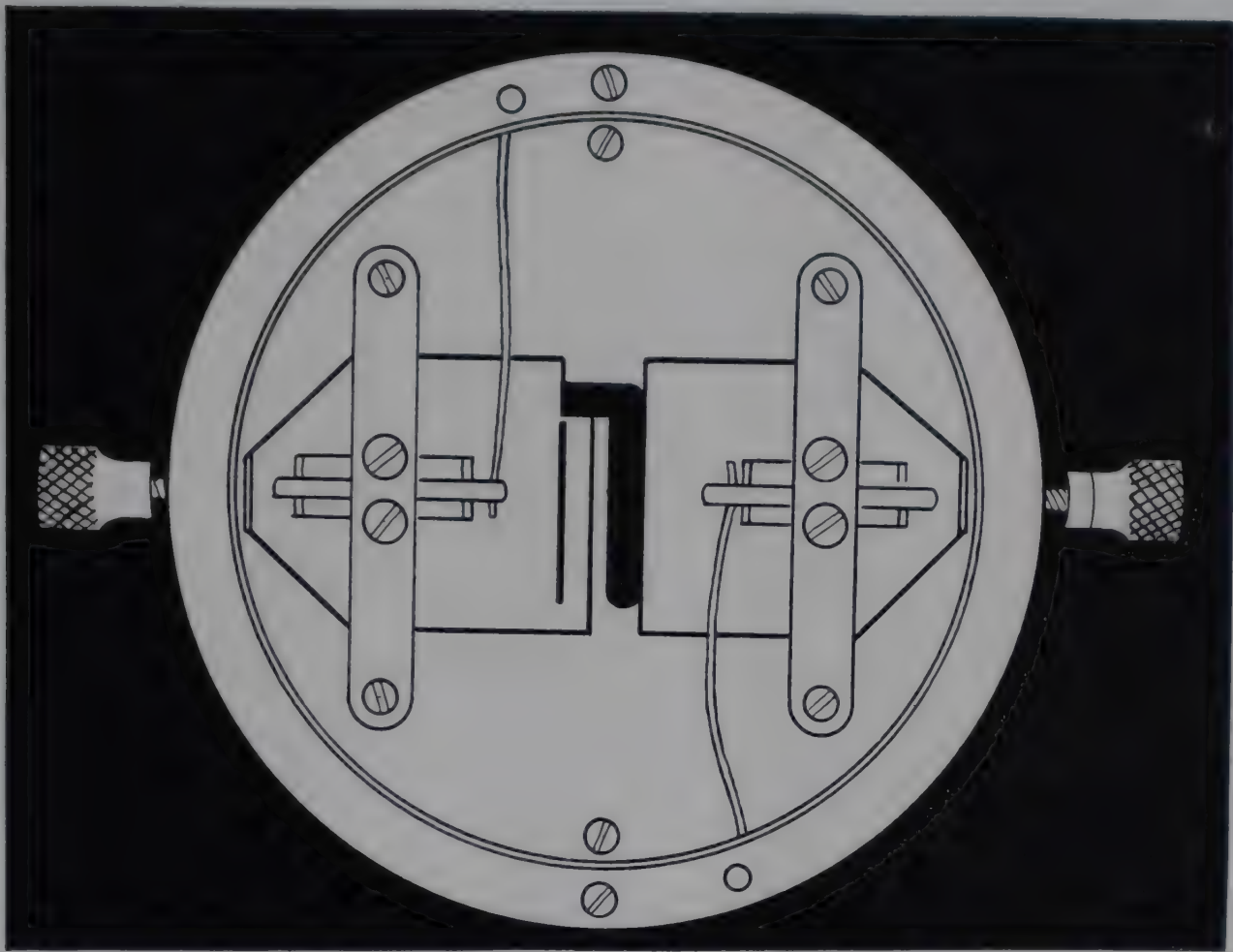


FIG. 17. The mechanism of the López-Lacarrère double slit. (After López-Lacarrère.)

KOEPPE'S TUBE

To prevent scattering of the emergent rays, Koeppe inserted a blackened tube (Fig. 9) between the diaphragm and the illuminating lens. In some models, there is a Rekoss revolving disk with color filters attached to the distal end of this tube.

A plano-convex cylinder of 9.0 diopters, axis 180 degrees, in the opening of the Koeppe tube was added by Koby.¹⁷³ By this method, the light is concentrated in the vertical axis. As a result, three times as much filament is used as a source of illumination. At the proximal focus of this beam, the luminosity is three times greater, while at the distal focus the intensity remains unchanged. However, the height of the beam is three times as large, permitting a greater area

of observation, including the entire cornea and lens. It is used with Vogt's method of illumination with low magnification.



FIG. 18. Corneal parallelepiped and optic section obtained with the double slit of López-Lacarrère.
(After López-Lacarrère.)

THE ILLUMINATING LENS

The illuminating lens is situated at the end of the arm nearest the patient. It is a biconvex lens with the less curved surface facing the patient's eye (3.05 D.). The other surface is aspheric (toward the observer) and has a greater curvature (about 11.00 D.). Thus

an aplanatic lens without spheric aberrations is obtained. Later, Vogt incorporated a smaller diaphragm aperture ($10/16$) to minimize the effects of aberration (Fig. 19). As the usual focal length



FIG. 19.



FIG. 20.

FIG. 19. Single illuminating lens of 7 or 10 cm. length with Arruga vertical adjustment and Elschmig focusing device. (Courtesy of Carl Zeiss, Inc.)

FIG. 20. Double rotating illuminating lenses with focal lengths of 7 and 10 cm. (Courtesy of Carl Zeiss, Inc.)

is 7 cm. (10×16 cm. diaphragm aperture), a very luminous and easily handled beam is produced; however, a lens of longer focal length 10 cm. (15×45 cm. aperture) is of advantage when using the arc lamp, since the shorter focal lens (7 cm.) does not include the entire crater image. The 10-cm. lens is also of advantage in simultaneously examining the zone of discontinuity in the lens and also for deeper penetration into the vitreous. Since the original illuminating lens was circular in shape and extremely large in diameter, it could easily collide with the microscope, especially when using a narrow angle between the illuminant and observation. To avoid this handicap, the present models use narrow rectangular lenses.

Some biomicroscopes (Fig. 20) are supplied with both lenses mounted on a single rotating support, so that they are rapidly interchangeable. The illuminating lens is mounted in a slot at the end of the arm in order that backward and forward motion may be obtained by use of rack and pinion screw for a distance of approximately 50 mm.; thus, it is possible to focus the beam accurately

on the plane of the eye under observation. To facilitate a more rapid vertical displacement of the lens and the beam, Arruga⁴ suggested a screw attached to the lower end of the illuminating lens.

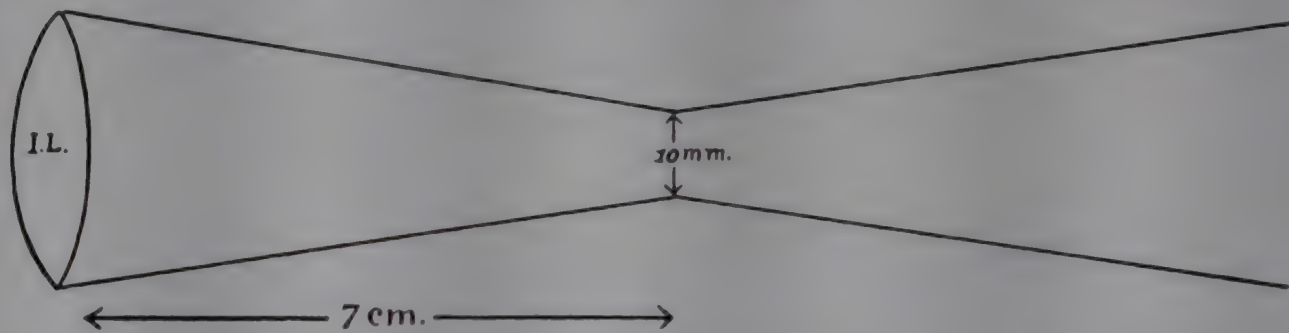


FIG. 21. Diagram of the beam emerging from illuminating lens. I.L., Illuminating lens.

Elschnig⁷⁸ devised a screw mechanism for combined horizontal and vertical movements.

OPTICAL CHARACTERISTICS OF THE LUMINOUS BEAM

The beam, which emerges from the rectangular illuminating lens, focuses at a distance of either 7 or 10 cm., depending on the power of the illuminating lens employed. The rays diverge from the point of focus. The beam resembles two pyramids, the apices of which are touching (Fig. 21). The width of the beam at its focus depends on the width of the slit; its outlines are sharp, and obviously the intensity of the illumination depends on the type of lamp used. Vogt has shown that with his method the beam is one and a half times more luminous than with Gullstrand's method, and that the height of the beam in its focal part is almost double.

TABLE I

VARIATIONS IN LUMINOUS INTENSITY OF THE
PRIMARY IMAGES

	VOLTS	AMPERES	CANDLES/CM ² .
Nernst	110	0.48	230
Pointolite	35	0.6	269
Nitra	10	3.9	1,335
Arc lamp	58	3.4	11,550

Hartinger,¹⁴⁶ in 1923, studied the variations in luminous intensity of the primary images derived from the Nernst, nitra, pointolite, and arc lamps by means of photometry. Employing the same optical apparatus for all the lamps he obtained the data in Table I.

He also calculated the brilliance of the focal part of the beam, using similar sources, interpolating lenses of 7 cm. and 10 cm. focal length, respectively (Table II).

TABLE II
LIGHT INTENSITIES OF VARIOUS SOURCES

	LENS OF 7 CM.	LENS OF 10 CM.
Nernst	48,000 lux.	18,800 lux.
Pointolite	56,300 lux.	22,000 lux.
Nitra (10 volts)	279,000 lux.	109,000 lux.
Arc lamp	2,417,000 lux.	945,000 lux.

In the focal part of the beam, greater luminosity is found with the 7 cm. than with the 10 cm. lens. As Koby pointed out, the homogeneity of the beam is as important as its intensity.

The differences between the methods of Gullstrand and Vogt can be easily demonstrated if beams projected by each system are studied in a completely darkened room with the light thrown against a flat surface. With the Gullstrand technique, a luminous ellipse is formed, many times the size of the illuminating lens (Fig. 23). Its borders show a variety of colors owing to the chromatic aberration of the condensing lenses. This scattering of light is not only wasteful, but also annoying to both subject and observer. The spirals of the filament are discernible in the focal part of the light, and the edges are not as sharp as in Vogt's method. With Vogt's method the light is concentrated on the surface of the illuminating lens, where the image of the filament is sharply outlined. In this way, the luminous ellipse is avoided, and the focal part of the beam of light is more homogeneous, containing no image of the filament (Fig. 25). The advantage of Vogt's method lies in the fact that the luminous in-

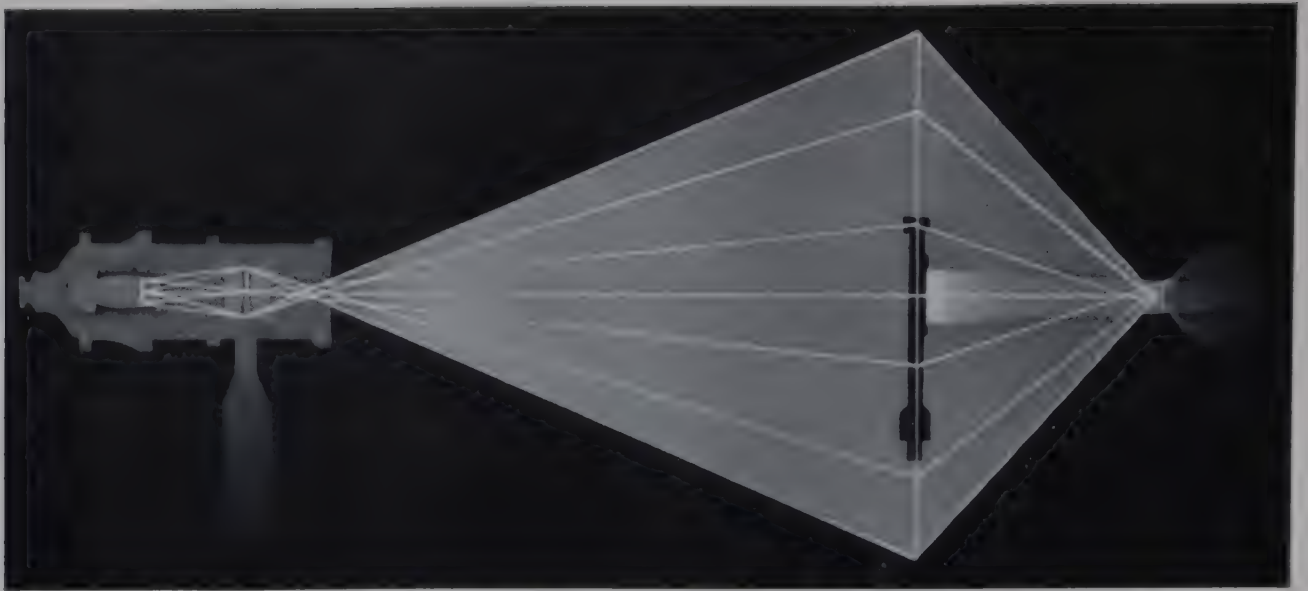


FIG. 22. Diagram of the projection of the luminous rays with Gullstrand technique. The rays from the light source are focused by the condensing lenses in the slit diaphragm, which sharpens the margins of the emergent beam by cutting out the peripheral rays. At the focal point of the illuminating lens an image of the filament is reproduced. Owing to the high degree of divergence only part of the light is gathered by the illuminating lens. (After López-Lacarrère.)



FIG. 23. A frontal section showing size of luminous ellipse in the plane of the illuminating lens with Gullstrand method of projection. Insert shows the size of the beam at the focal point of the same illuminating lens. (After López-Lacarrère.)



FIG. 24. Diagram of projection of the luminous rays according to the Kohler-Vogt technique.
(After López-Lacarrère.)



FIG. 25. Frontal section of luminous beam in the plane of the illuminating lens (Vogt's method of projection) at its focal point. The image of the filament is clearly outlined on the diaphragm of the illuminating lens, having only a small degree of chromatic aberration, clearly visible in the incandescent spirals. All the light is projected on the diaphragm of the illuminating lens and consequently there is no scattering of light as with the Gullstrand method (cf. Fig. 23). In the insert, it is seen that the focal part of the light is more homogeneous than in Gullstrand's method because it does not contain the image of the filament. Chromatic aberration is minimal and its luminous intensity is greater. (After López-Lacarrère.)

tensity of the beam is greater and the chromatic aberration at its borders minimal. The disadvantages in the Gullstrand method have also been corrected in the newer models.* A fine-grained ground-glass disk in front of the condensing lens diffuses the image of the spiral filament of the lamp bulb producing an even illumination of great intensity (page 40).

THE STEREOSCOPIC MICROSCOPE

As previously mentioned, many attempts were made to examine the living eye under magnification long before the biomicroscope was devised. The "Manuel d'ophtalmologie pratique" by de Wecker and Masselon contains a picture showing the application to this purpose of a monocular microscope with a lens for oblique illumination. The monobjective binocular microscope, introduced by Abbé in 1881 and further modified by Seidentopf in 1885, was the first binocular instrument employed. A stereoscopic effect was obtained by splitting the light from the single objective with a double system of prisms, which provided an image for each ocular (Fig. 26). However, this system results in loss of light because of absorption and halving of the original intensity. This disadvantage is accentuated, if the Porro system of erecting prisms is added. Koeppe has employed this type of microscope for fundus visualization (orthobitumi of Zeiss). The monobjective monocular microscope is utilized in advanced spectroscopic and polariscopic studies of the eye.

Before biomicroscopy was devised, the binocular corneal microscope (Czapski) was used with the Lucanus curved rail under the microscope; this rail, quadrant in shape, held a movable illuminating device (an ordinary lamp of 4 volts, 0.4 ampere). One side of the microscope was available for manipulations on the eye and with the quadrant in a vertical or oblique position the light may be directed upward. The curvature of the rail was such that the optic axis of the illuminating device and that of the microscope were always directed on the same point so that the part under observation might

* Bausch & Lomb Optical Company.

be well illuminated from any position (Fig. 2). This produced a concentrated and more or less diffuse patch of light on the eye similar to that obtained by oblique illumination. But, as already

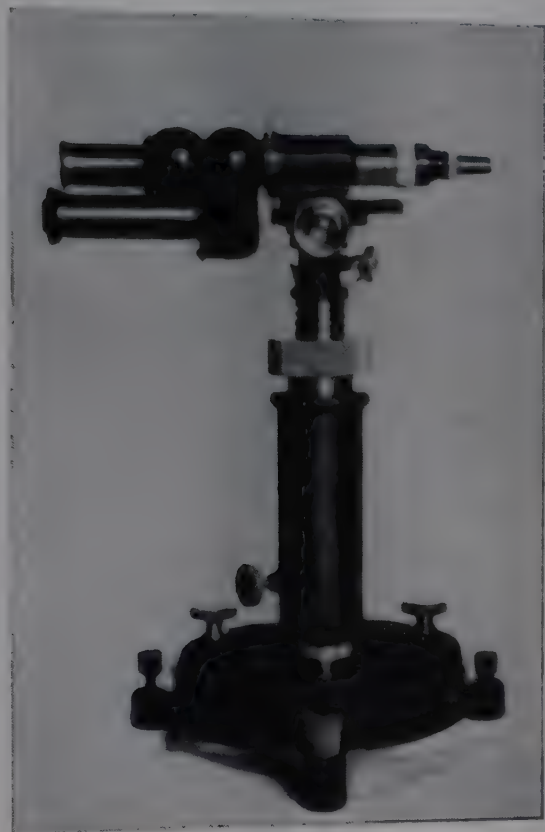


FIG. 26

FIG. 26. Bitumi with microtubus. (Courtesy of Carl Zeiss, Inc.)

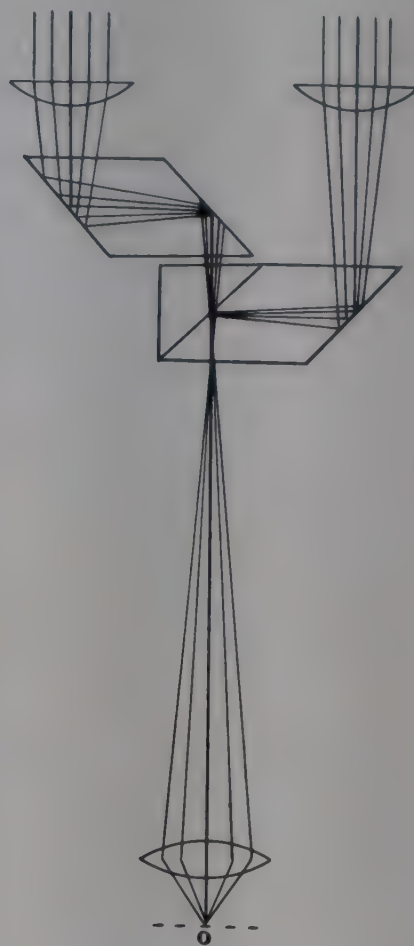


FIG. 27

FIG. 27. Diagram of light pathways in orthobitumi microscope. Entering beams are split by prisms so that each eyepiece receives half the beam.

pointed out, this apparatus did not embody the system of maximum focal illumination, hence could not yield the results obtained by the later Gullstrand-Vogt and Koeppe techniques.

All modern biomicroscopes utilize the binocular microscope. This microscope was designed by Czapski and Schatz⁵⁴ following the ideas of Aubert⁵ and Greenough* (Figs. 28, 29, 30). The Czapski binocular microscope consists of three main parts: (a) the eye pieces or oculars; (b) the objectives, and (c) a system of Porro prisms

* Horacia S. Greenough was an American biologist who worked in Paris. He designed a microscope of this type about 1892. Czapski and Schatz modified it in 1897. The binocular microscope with two objectives and two oculars is in principle like the original binocular telescope prisms so that each eyepiece receives half the beam.

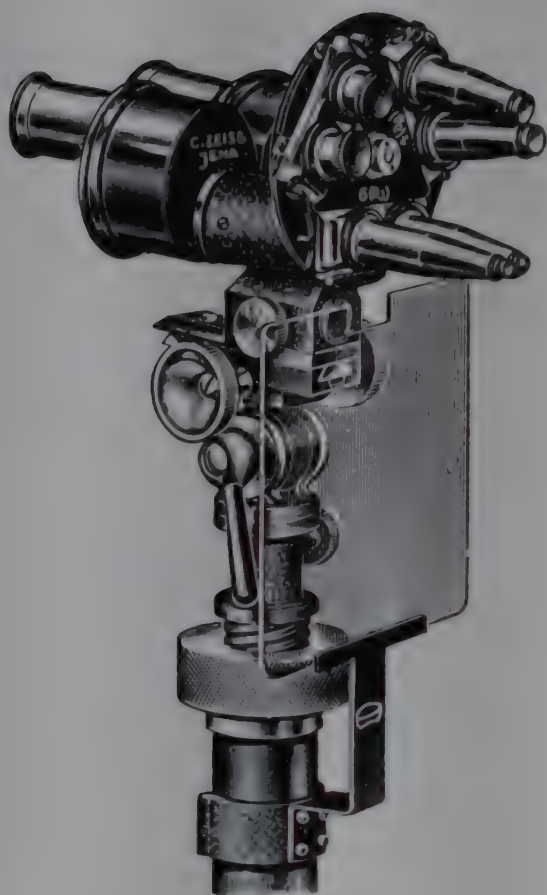


FIG. 28

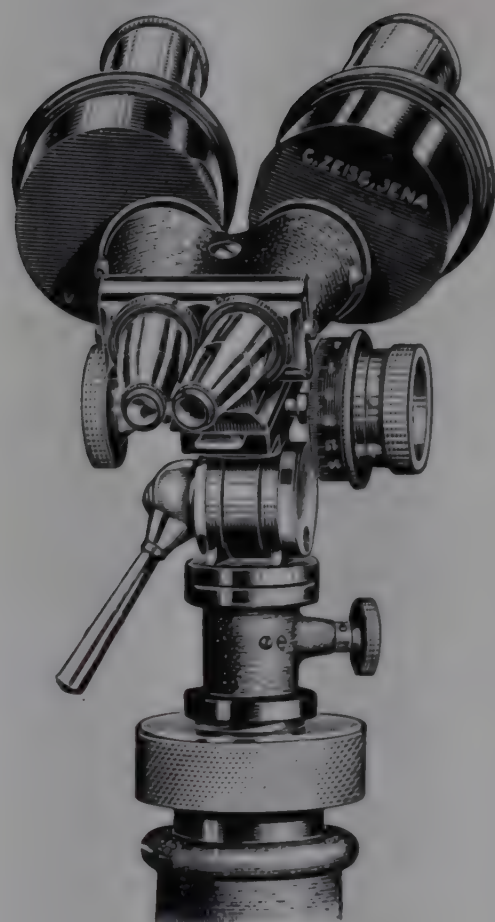


FIG. 29

FIG. 28. Czapski binocular microscope with revolving objectives. (Meesmann, A. Die Mikroskopie des lebenden Auges an der Gullstrandschen Spaltlampe mit Atlas typischer Befunde. Courtesy of Urban & Schwarzenberg.)

FIG. 29. Corneal microscope (Czapski) with Ulbrich drum micrometer for measuring displacement. (Courtesy of Carl Zeiss, Inc.)

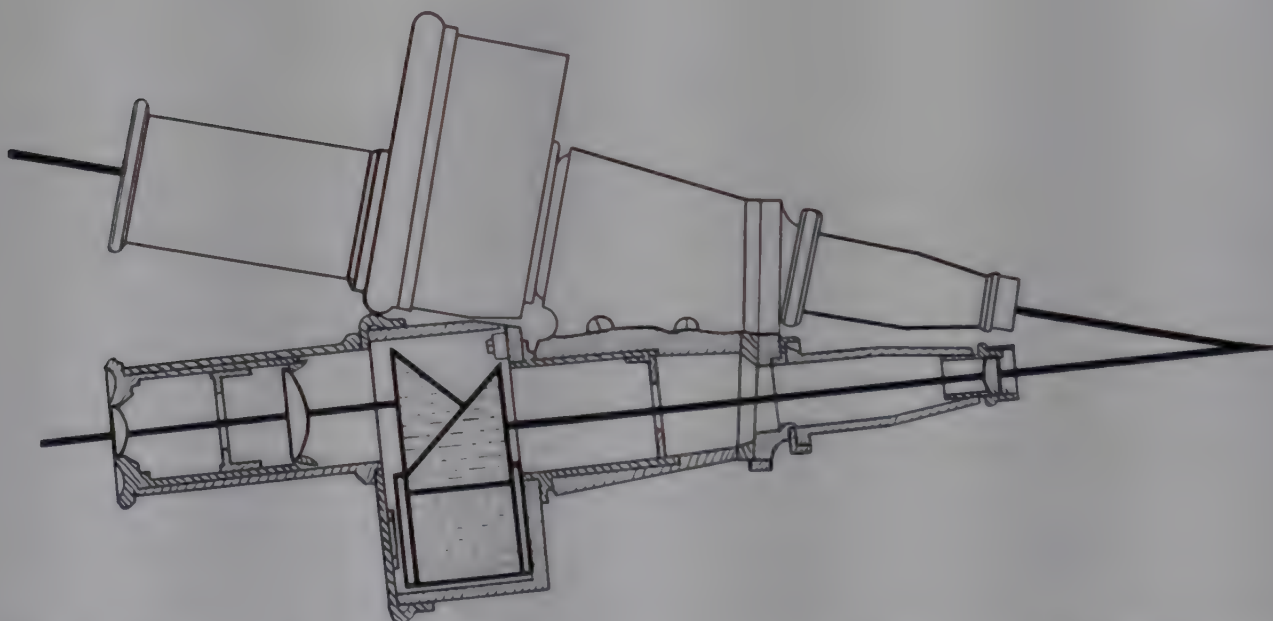


FIG. 30. Diagram of optics of Czapski corneal microscope. (Meesmann, A. Die Mikroskopie des lebenden Auges an der Gullstrandschen Spaltlampe mit Atlas typischer Befunde. Courtesy of Urban & Schwarzenberg.)

with four reflecting surfaces. This microscope affords an erect stereoscopic image. Each ocular slips into a tube which is attached to the prism housings. The two objectives are mounted on a metal plate (21 by 50 cm.) and converge at a 14 degree angle. The tubes containing the oculars and the drums are adjustable to conform with the interpupillary distance of the observer.

CZAPSKI MICROSCOPE (ZEISS MODEL)

With the Czapski microscope three objectives, known as F. 55, a 2, and a 3, can be used interchangeably. Their respective focal distances are 55 mm., 37 mm., and 28 mm. The oculars * are numbered from 1 to 6; numbers 2, 4, and 6 are the ones usually employed. When changing both objectives and oculars in the dark it is helpful to remember that the length of the objectives increases with their power; the reverse is true of the oculars, which become shorter as their strength increases.

The apparent steadiness the eye maintains during fixation results from a continuous muscular activity, which under ordinary conditions escapes attention. By closer observation, fixation is seen to be composed of three types of movements (Duke-Elder): (1) relatively large jerky movements; (2) rapid, fine excursions in the intervals between the larger jerky movements; (3) minute movements of the head.³¹⁹

When the higher magnifications are used (over 40 ×) these movements present practical difficulties. With increased power the involuntary physiologic oscillations of the eyeball become more manifest and accurate observation is difficult. In addition, the closeness of the high-powered long objective to the patient's eye is disturbing.

Using various combinations of these Zeiss oculars and objectives it is possible to obtain magnifications varying from 8 × to 103 × as shown in Table III.

* The oculars are of the Huygenian type, i.e., a combination of two plano-convex lenses, which are usually employed with achromatic objectives.

TABLE III
TABLE OF MAGNIFICATIONS (ZEISS)

OCULARS	OBJECTIVE F. 55	OBJECTIVE a 2	OBJECTIVE a 3
No. 1	8	20	31
No. 2	9	23	35
No. 3	13	32	50
No. 4	16	40	61
No. 5	23	57	88
No. 6	26	67	103

Practically every detail observed by means of biomicroscopy is discernible with $23\times$ or $35\times$ magnification. These magnifications can be obtained with the No. 2 eyepieces * and the objectives a 2 and a 3. It is my opinion that a magnification of $23\times$ is the most generally useful. This magnification can be obtained in certain instruments † by using the F. 55 objectives, combined with $15\times$ wide-angle eyepieces. In most of the modern instruments there is a battery of two or three objectives, fitted on a revolving disk (Pardo).

The advantage that the Czapski-Zeiss or Greenough microscope has over other instruments (e.g., Abbé, Bitumi [Seidentopf], ortho-bitumi) lies in the fact that this instrument has two independent optical systems, which produce images equal in size and luminosity for each eye. In the other microscopes with single objectives, the image is split by prisms in the ocular system, and thus stereoscopic vision is obtained at the expense of brilliance.

The Czapski microscope is mounted on a horizontal slide for sagittal displacement of 3.5 cm., which is controlled by twin-

* A disadvantage of high power eyepieces is that owing to the very small eye points, minute specks of dust or dirt on the lenses or on the surface of the eyes are apparent in the field of view. Small particles may obstruct a relatively large portion of the eye point. In actual practice the eye itself, as a result of its dioptric properties (Fig. 31), adds to the above magnifications. This increase in power becomes greater as the examination progresses from the cornea to the vitreous. For example, in observing a deposit on the posterior surface of the lens it is necessary to multiply the outside magnification used by 1.7 in order to obtain the real magnification.

† Bausch & Lomb Optical Company.

milled screws. Attached to the under surface of this horizontal slide, is a joint for the purpose of tilting the instrument forward or backward. The vertical support, extending downward from this joint,

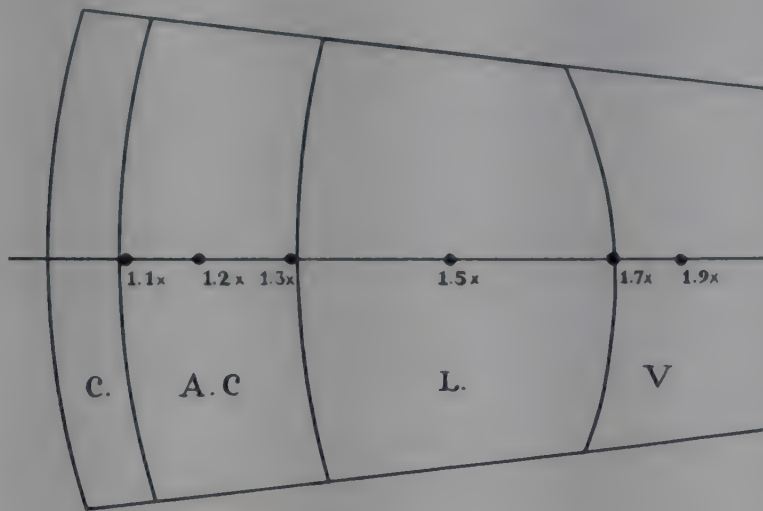


FIG. 31. Magnification factors at various levels within the eye resulting from its intrinsic dioptric power. The numbers indicate the amount by which the magnification afforded by the microscope must be multiplied to indicate the actual magnification at various positions in the eye. C, cornea; A. C. anterior chamber; L., lens; V, vitreous.

is so constructed that the microscope can be rotated on its vertical axis and fixed in any position. These parts are mounted on a strong vertical support, which has a helical screw, and permits raising and lowering the microscope for a distance of 5.5 cm. The base of the vertical support fits into a large circular disk. The disk can be fitted into either a movable compound slide or an annular tripod for use on a glass-topped table; in the latter case it rests on three feet, and can easily be moved by hand. I prefer the latter because of the ease and facility with which the entire microscope can be moved. The glass plate covering the table must be kept scrupulously clean in order to ensure the smooth sliding of the instrument over its surface. The table on which the microscope rests can also be raised or lowered. Under the base of the support of Henker's arm (on which the various parts of the illuminating system rest), a horizontal articulating arm is attached. This arm is joined to the upper end of the support of the entire table. Because both the chin-rest and the lamp are attached to this table, the vertical adjustment raises the entire apparatus with a single motion.

The original Zeiss instrument has been described in detail not only because it was the first available but also because it solved many of the optical and mechanical problems connected with bio-

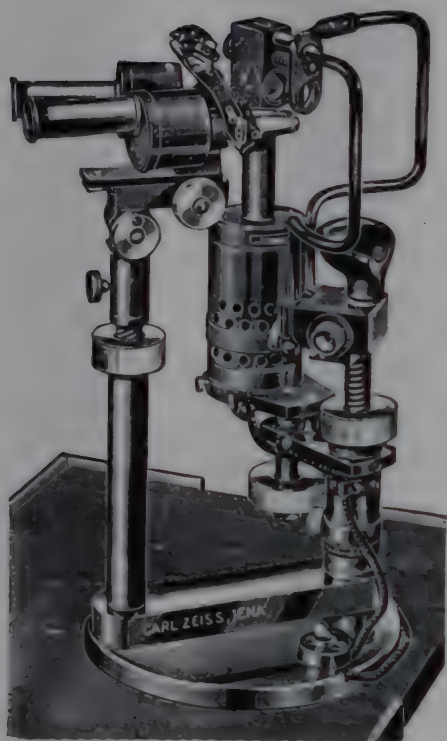


FIG. 32

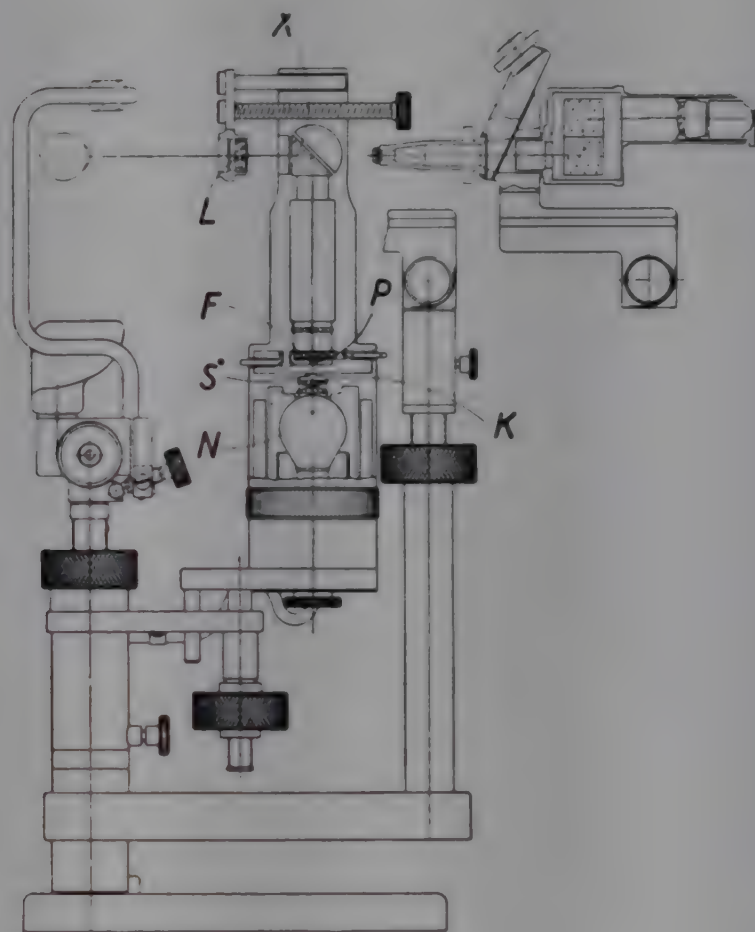


FIG. 33

FIG. 32. Biomicroscope after Comberg. (Courtesy of Carl Zeiss, Inc.)

FIG. 33. Cross section of biomicroscopic equipment (after Comberg). For the sake of clearness the body part of the corneal microscope is shown separately at the right. N, Nitra bulb; K, condenser; S*, slit; P, Rekoss disk with diaphragms; F, Rekoss disk with red absorbing filter and gray glass filter; X, prism; L, illuminating lens. (Courtesy of Carl Zeiss, Inc.)

microscopy. However, the instrument had a number of faults. For example, the floating articulations of the long arm of the illuminating system proved awkward, because at the slightest movement of the observer or even if the floor was slightly irregular, they moved out of position. The change required in the position and angle of the arms of the illuminating system, when shifting from right to left and vice versa, was most inconvenient, because the observer had to draw away from the table in order to swing the arm. In the meantime the patient might have moved his head and a tiresome re-

adjustment had to be made. Such defects led to modification of the older models.



FIG. 34. Biomicroscopic equipment (Comberg) ready for use. (Courtesy of Carl Zeiss, Inc.)

COMBERG SLIT LAMP

In 1936, Comberg devised an improved model of the slit lamp (Figs. 32, 33). The chief modification in his instrument consisted in shifting the illuminating system from a horizontal to a vertical position. The vertical arm is considerably shorter than the original horizontal arm of Henker; this change is made possible by substituting lenses of 41 D. for the two 11 D. condensing lenses. Simply turning a sleeve on this apparatus changes a circular field of illumination to a slit; by continuing the same motion, this slit can be narrowed until it is completely obliterated. At the lower end of the vertical arm is a nitra bulb. Above this two Rekoss disks are housed; one contains colored filters and the other has two stenopeic holes for the production of cylindrical beams. At the upper end of

the vertical arm is a rectangular prism which turns the beam in a horizontal direction. The horizontal beam then passes through the illuminating lens which has a focus of 7 cm. In this way, the image of the filament is projected on the illuminating lens by means of a rectangular prism. The microscope and the illuminating system are

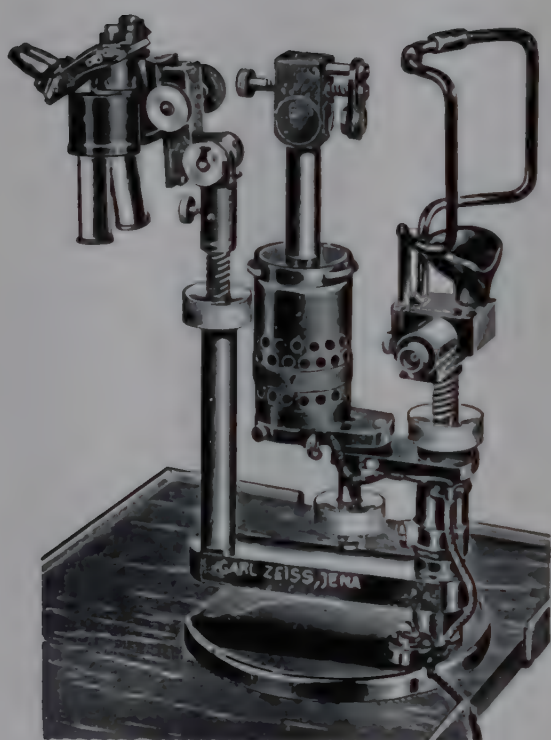


FIG. 35

FIG. 35. The microscope may be tilted back to permit the lamp to be shifted from side to side. (Courtesy of Carl Zeiss, Inc.)

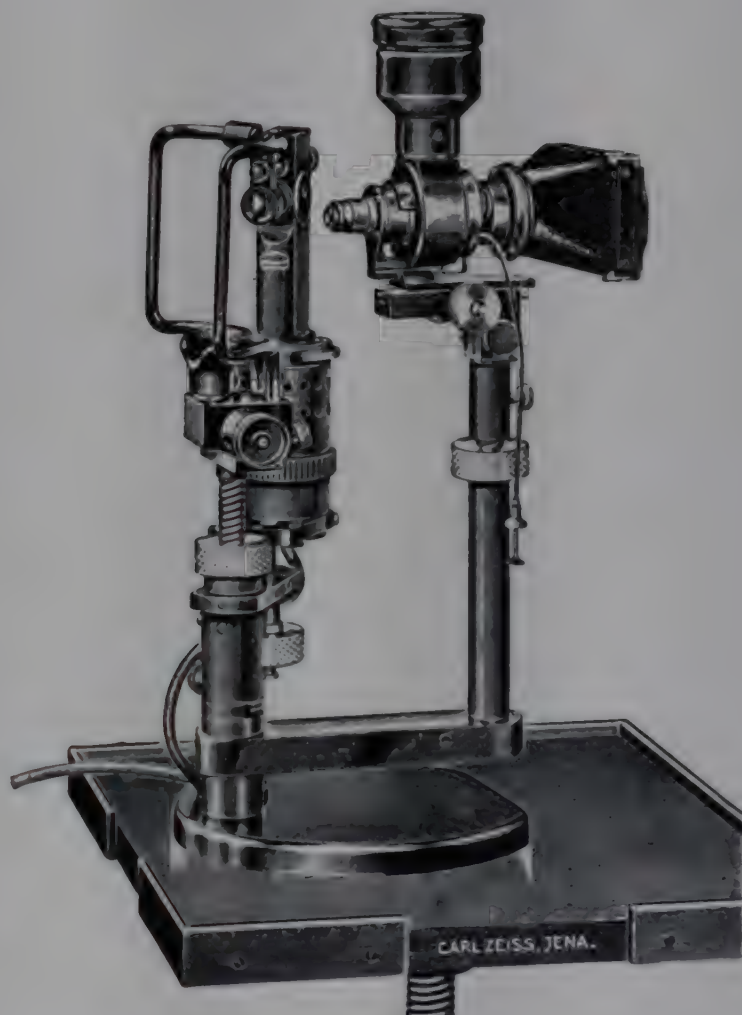


FIG. 36

FIG. 36. Reflex camera attachment for biomicroscopic photography. (Courtesy of Carl Zeiss, Inc.)

attached to a pillar which supports a chin-rest. The apparatus is constructed so that the microscope and illuminating system may be rotated either individually or together; the entire apparatus can be moved without altering the angle between them (Figs. 34, 35, 36). Moreover, the support for the head, as well as the chin-rest, can be moved laterally so that different areas of the eyes may pass through the field of observation, in a manner similar to that in which objects are moved on the stage of a laboratory microscope.

UNIVERSAL SLIT LAMP * (KOEPPPE MODEL)

This instrument, although it incorporated many novel ideas, proved cumbersome in the hands of beginners. The multiplicity of

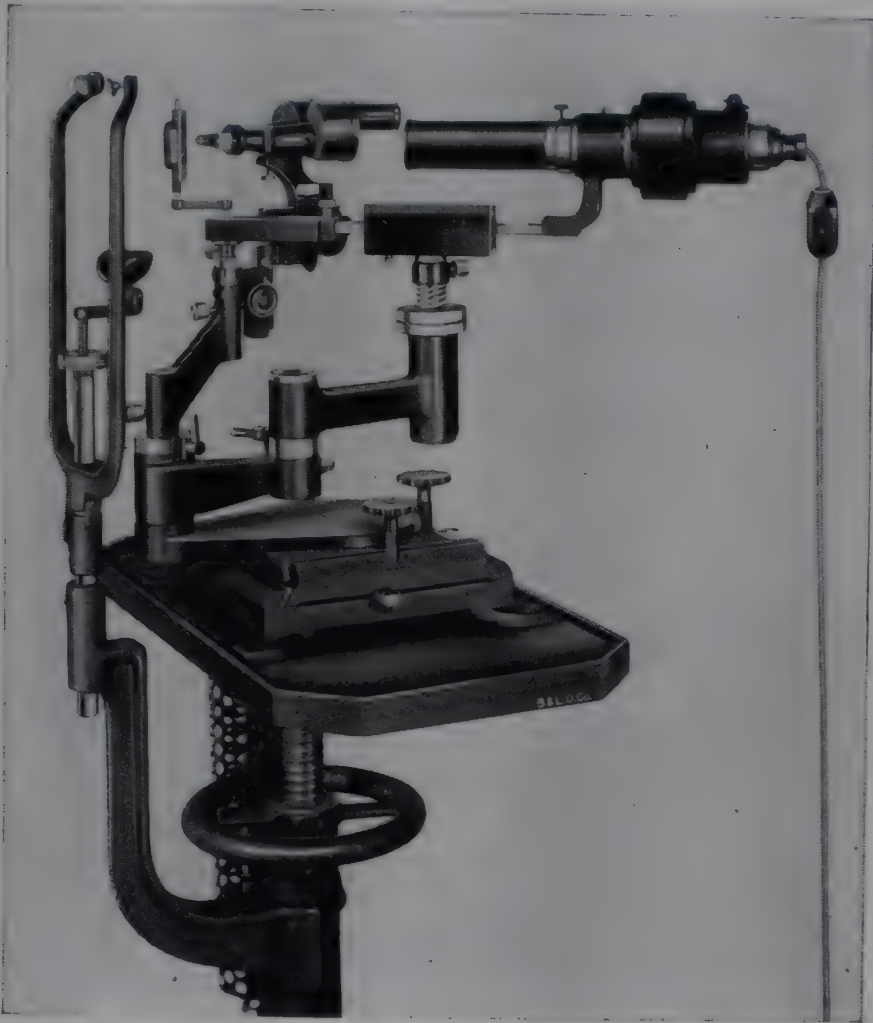


FIG. 37. Koepppe universal slit lamp. (Courtesy of Bausch & Lomb Optical Company.)

mechanical adjustments necessary to operate the instrument militated against its efficient use (Fig. 37).

The illuminating system and the binocular microscope were mounted on a heavy compound slide, so that their common vertical axes permitted concentric rotation of the supporting arms. The vertical axis ran approximately through the center of the rotation of the examined eye. After focusing and locking the adjustments, the microscope and the illuminating system could be rotated without disturbing their focus.

The binocular microscope was of the Greenough type, having

* Bausch & Lomb Optical Company.

converging oculars and paired objectives. The oculars were of $6.4\times$ and $10\times$; the objectives 55 mm. and 32 mm. With these combinations magnifications of $13\times$, $20\times$, $36\times$ and $56\times$ could be obtained.

The source of illumination was a 19 volt, 2.5 ampere mazda lamp with a straight helical filament. A variable transformer provided proper reduction of the ordinary 110 volt alternating current. The image of the source was projected on an adjustable slit by a condensing system (Koeppé), employing lenses of 50 D. A diffusing glass obliterated the filament and the rays were then collimated or made parallel by another — intermediate — lens before striking the illuminating lens. Hence, no image of the filament (comparable to the Vogt method) was formed on the surface of the illuminating lens, but there was an image of the slit, which, when properly focused, appeared as a brilliant rectangle of illumination. This system of illumination is still used in all the later models.* It results in a brilliance about one and a half times greater than that of the nitra lamp used with the Kohler-Vogt method of illumination.

SIMPLIFIED UNIVERSAL SLIT LAMP †

Following the Koeppé model of the universal slit lamp an improved simplified model, designated the "Universal slit lamp," was made in 1928 (Figs. 38, 39, 40). In this model, the heavy mechanical compound slide of the Koeppé universal model was eliminated. Instead, the binocular microscope was mounted on a heavy metal pedestal base, which could easily be moved on the glass surface of the instrument table. A rack and pinion vertical adjustment was provided for the binocular microscope. A long articulated arm fastened the illuminating system to the table; this could be swung from side to side for either eye. The Koeppé system of illumination was employed, the source of illumination being a 10 volt, 2.5 ampere

* Bausch & Lomb Optical Company.

† Made by Bausch & Lomb Optical Company.



FIG. 38. Simplified universal slit lamp. (Courtesy of Bausch & Lomb Optical Company.)

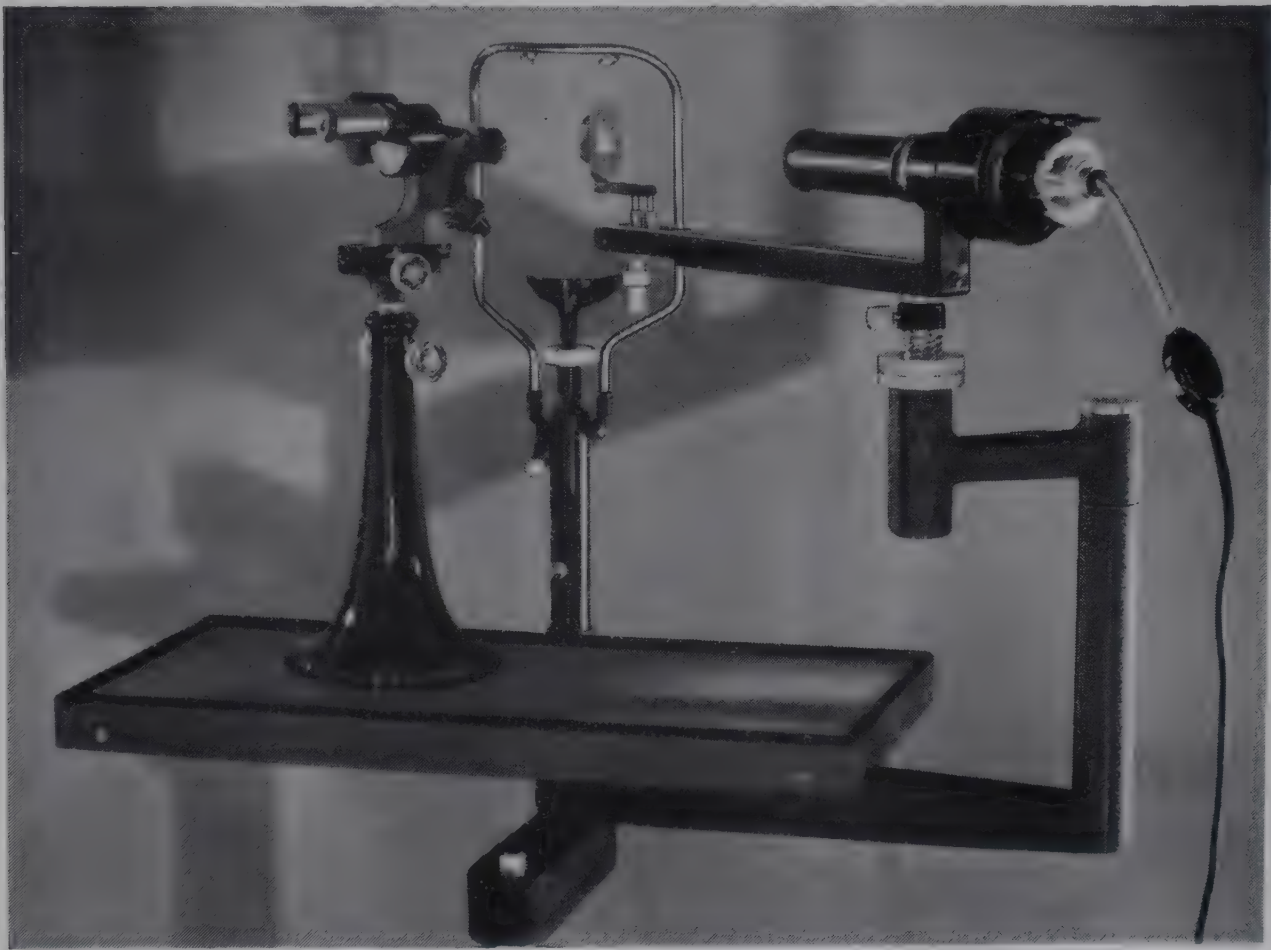


FIG. 39. Universal slit lamp illumination assembly. (Courtesy of Bausch & Lomb Optical Company.)

lamp with a straight helically coiled filament. This filament was projected by the condenser lenses *C* (about 46 D.) on a slit *D* in Figure 40. A diffusing glass *R* was used to break up the filament

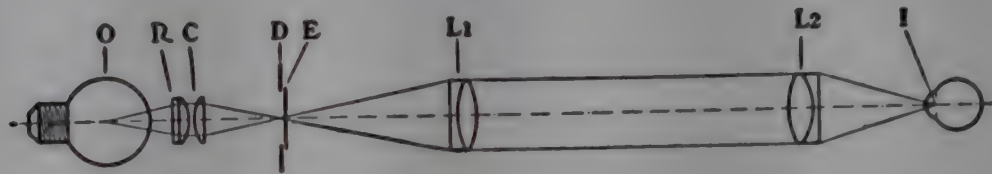


FIG. 40. Diagram of Universal (Koepe) illumination system. *C*, Condenser system; *D*, slit; *E*, diaphragm disk; *I*, patient's eye; *L*₁, intermediate lens; *L*₂, illuminating lens; *O*, lamp; *R*, diffusing disk. (Courtesy of Bausch & Lomb Optical Company.)

structure in the image. The slit *D* can be varied in width from 1.7 mm. to the thinnest usable beam by means of a screw. The length of the slit is changed by means of the circular plate *E* carrying apertures of the following sizes: 7 mm., 3.5 mm., 1.7 mm. and 0.5 mm. By rotating the proper aperture before the slit, the length of the slit can be altered. These adjustments provide different sizes of beams and two circular pencils of illumination. The light, passing through the slit, is collimated by a lens, *L*₁, and an image of the slit is formed by the lens, *L*₂, which corresponds to the ordinary illumination lens. In the Koepe illumination system, as used in the Universal slit lamp, the collimating lens in front of the slit diaphragm is a doublet of total power of approximately plus 10.00 D. The objective focusing lens nearest the patient's eye is a plus 14.28 D. lens of 70 mm. focal length. It should be termed a diaphragm achromatic doublet with a convex portion of glass having a low refractive index (crown), and a concave portion of glass with a high refractive index (flint). To facilitate focusing of the illuminating lens, an Arruga screw mounting is provided, which gives a large range of vertical and horizontal movement of the slit beam for full examination of the eye. To adjust the lamp the lock nut is loosened and the lamp housing pushed in as far as it will go. A piece of white paper is held in front of the lens *L*₂. On the paper will be formed a hazy oval spot of light with a brighter streak running through it. This bright streak is an out-of-focus image of the

filament. The lamp housing should be rotated about a horizontal axis until this bright streak is vertical. By means of a screw the entire lamp housing can be shifted sidewise until the bright streak seen on the paper is centered vertically through the lens L 2. The lamp housing is now pulled outward without rotation until the oval spot is evenly illuminated, and at this point the wing nut is tightened. The spot of light, when the lamp is properly adjusted, will have a slightly speckled appearance. This is due to the diffusing glass which is to break up the filament structure, and will not be visible in the slit image.

The intensity of the illumination can be increased or decreased by means of the slide on the rheostat or, if a transformer is used, by rotating the voltage adjusting knob.

It sometimes happens that particles of dirt get on the jaws of the slit and will be seen in the slit image, particularly when the examiner is using a narrow beam. The slit can be cleaned by removing the entire unit from the lamp house casting and taking off the lens tube holding L 1, which allows access to the slit itself.

A variable transformer is employed for proper reduction of the ordinary 110 volt alternating current. There are two ranges, from 12 to 19 volts and from 19 to 25 volts, which give corresponding low and high intensities of light.

The binocular microscope (Figs. 41, 42, 43) is provided with a revolving nosepiece for two parfocal, paired objectives. The low power objective is a pair of 55 mm. lenses, while the high power objective is a pair of 40 mm. lenses. These can be combined with either a 10 × or a 15 × eyepiece * to produce various magnifications as shown in Table IV.

POSER BIOMICROSCOPE

Poser † designed a new instrument, incorporating many improvements. This instrument was designed, primarily, to overcome the

* These eyepieces are of the wide-field type (a positive type) and it is not possible to use the Huygenian eyepieces unless the microscope is readjusted.

† Bausch & Lomb Optical Company.

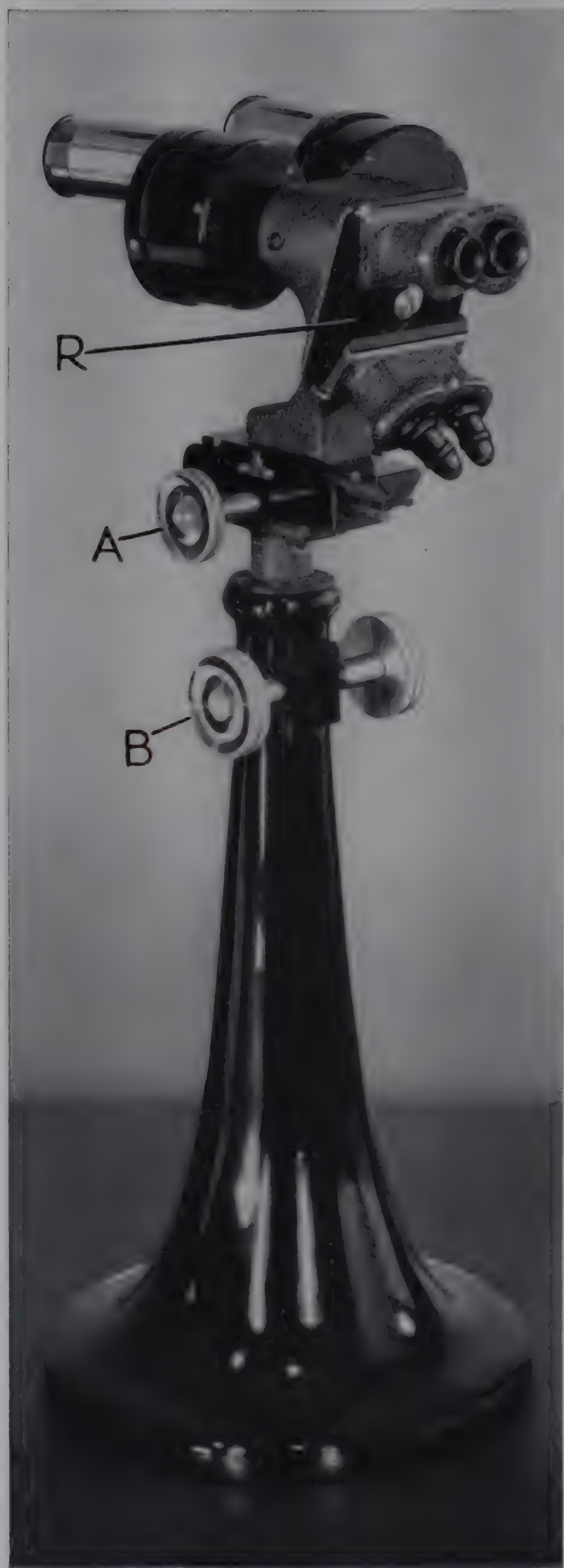


FIG. 41. Wide angle binocular microscope. A, The focusing button; B, elevating device; R, revolving nosepiece. (Courtesy of Bausch & Lomb Optical Company.)



FIG. 42. Binocular wide field microscope with Cowan magnification feature; Revolving nose-piece enables quick interchange between low and high powers. (Courtesy of Bausch & Lomb Optical Company.)

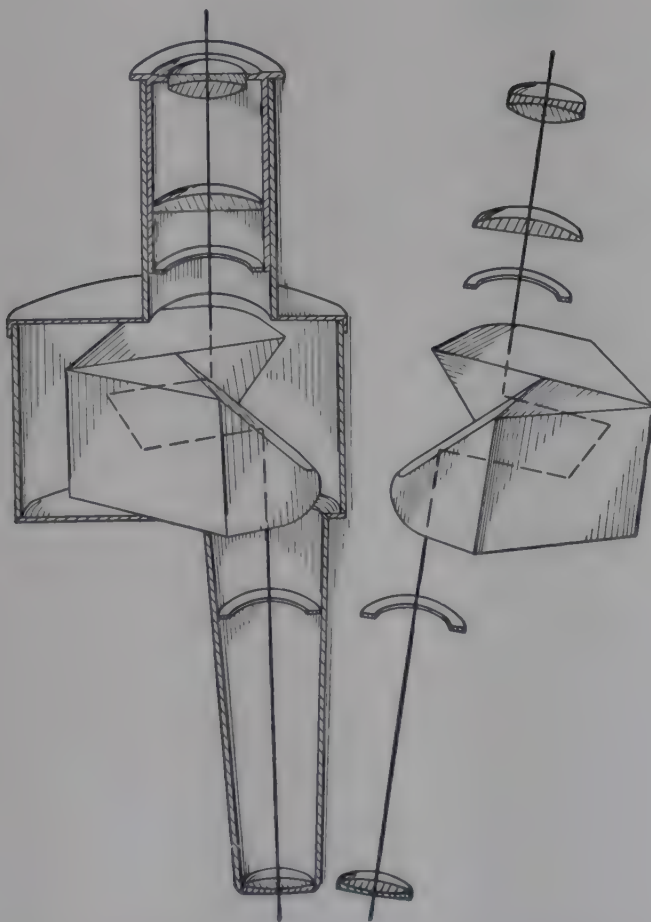


FIG. 43. Dissected view of Bausch & Lomb binocular microscope showing arrangement of lenses and prisms. (Courtesy of Bausch & Lomb Optical Company.)

TABLE IV
MAGNIFICATIONS OBTAINED BY COMBINATIONS
OF PAIRED OBJECTIVES WITH 10 × AND 15 ×
EYEPIECES

PAIRED OBJECTIVES	PAIRED WIDE-ANGLE EYEPIECES	
	10 ×	15 ×
55 mm. { Magnification	15.5 ×	22.5 ×
{ Width of Fields	12 mm.	10 mm.
40 mm. { Magnification	31 ×	47 ×
{ Width of Fields	5.5 mm.	4.4 mm.

obstacles often encountered in previous designs, such as lack of rigidity, length of illumination arms, and awkwardness in changing the direction of the beam from one eye to the other (Figs. 44, 45). It consists of a rigid base fitted with a longitudinal slide movement. There are double roller bearings, which insure a free movement of the instrument from one eye to the other. An automatic arresting device is provided for this movement so that the illuminating system and the binocular microscope can be fixed in any position. The horizontal slide of the instrument base is fitted with a strong pivot in the center; this serves as a vertical axis to which the illuminating system and the microscope are attached so that these may be independently rotated around this common axis. The pivot is in a vertical plane coincident with the center of rotation of the human eye.

The arm carrying the microscope is hinged at its lower end so that the microscope can be tilted up out of position, permitting the support of the illuminating system to be moved freely to the right or left eye of the patient. This facilitates rapid examination of either eye from the nasal or temporal side, as may be desired. There is a clamping device at the lower end which permits free rotation of the entire apparatus as a unit once the angle between the microscope and the illuminating system is fixed. When the clamping device is re-

leased, the illuminator and the binocular microscope can be rotated separately.

In addition, there is an elevating mechanism provided with a

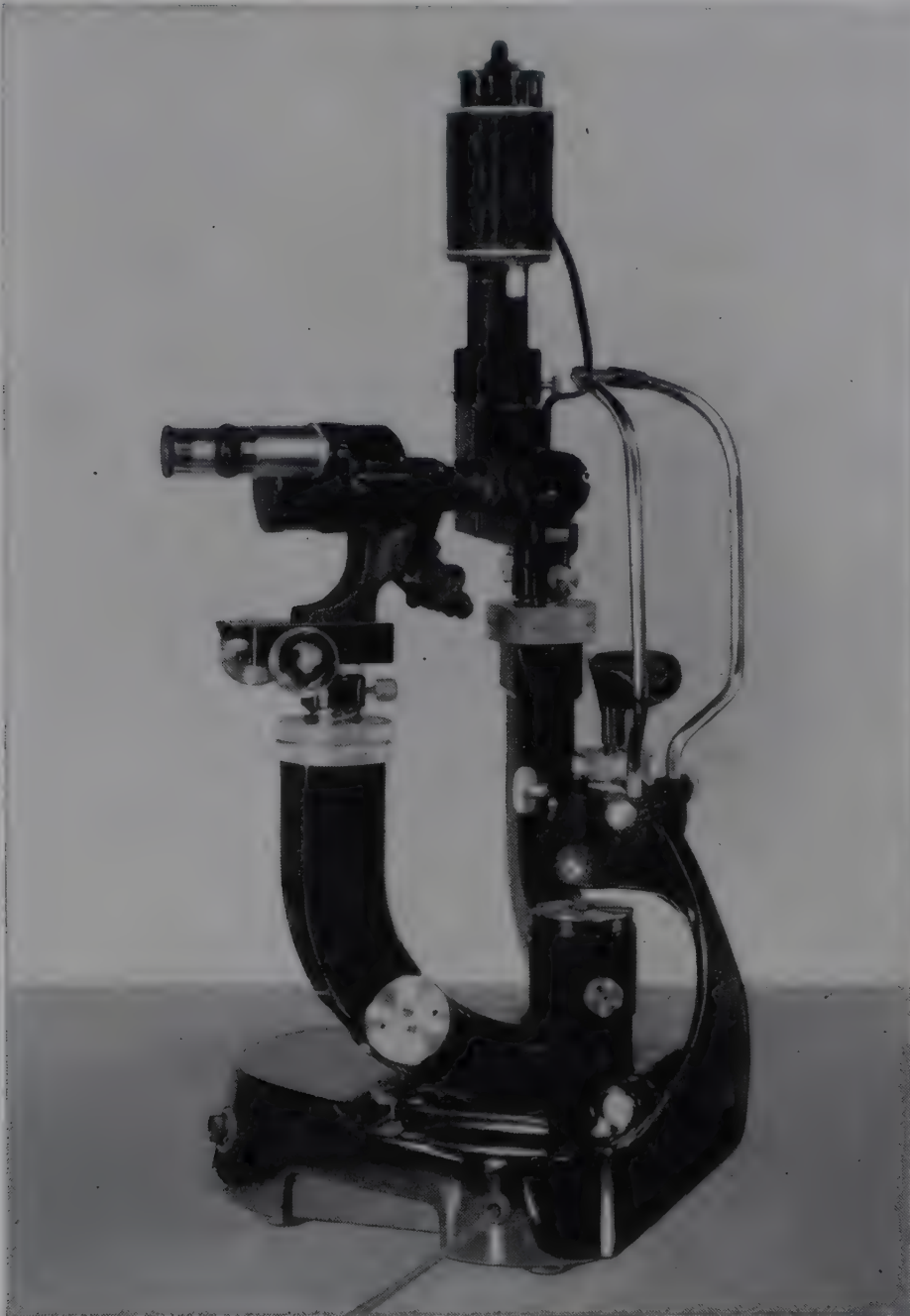


FIG. 44. Frontal view of Poser model of Bausch & Lomb slit lamp. (Courtesy of Bausch & Lomb Optical Company.)

socket and pivot joint, as well as an arresting device, so that the illuminating system can be secured in any desired position. The illuminating lamp, condensing lens system, adjustable slit, and rotating diaphragm disk are arranged in a vertical position. The aplanatic illuminating lens system is provided with a fine rack and pinion focusing device and is placed in a horizontal position.

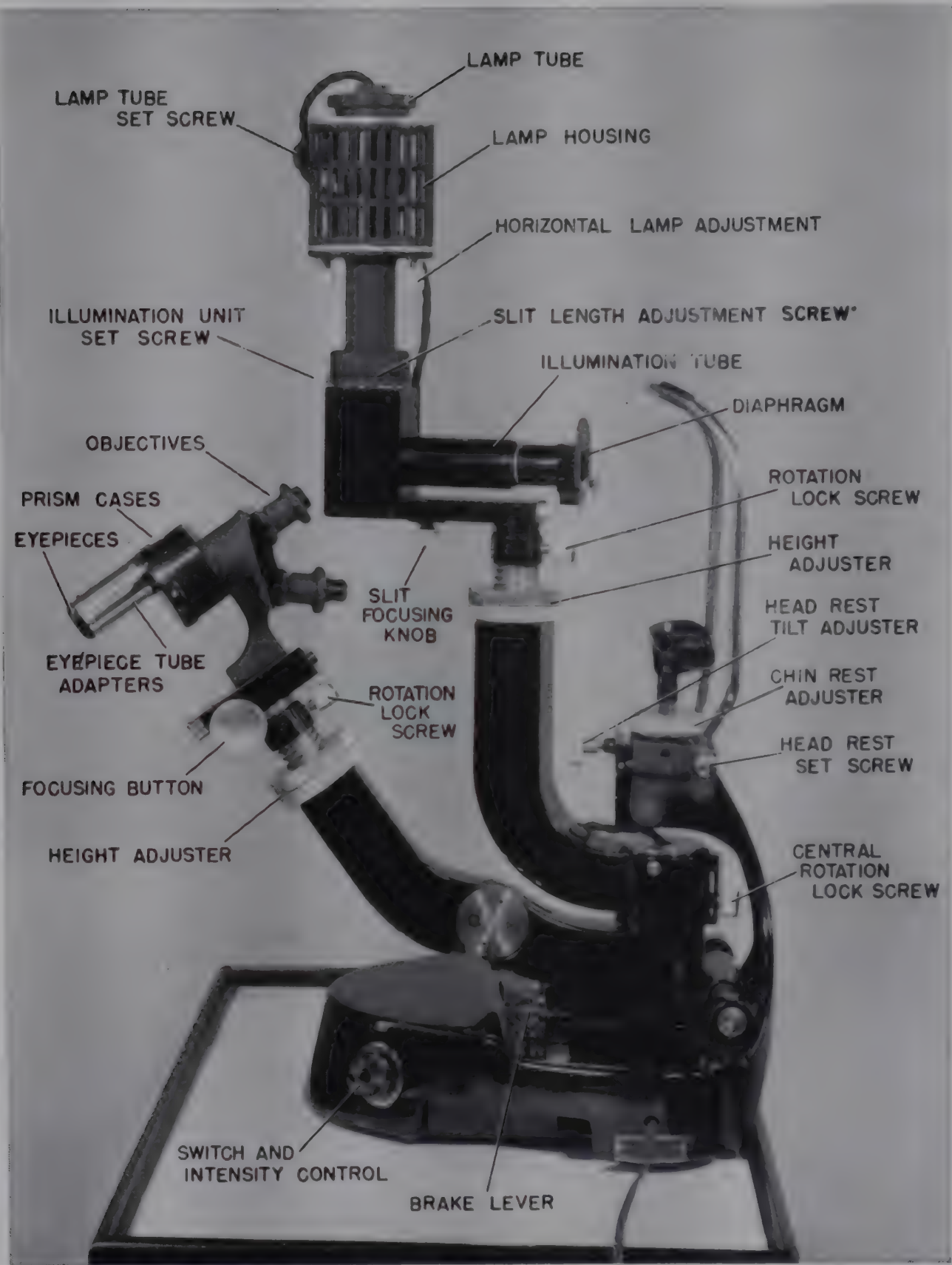


FIG. 45. Side view of Bausch & Lomb-Poser slit lamp. (Courtesy of Bausch & Lomb Optical Company.)

ILLUMINATING SYSTEM

The light source (Fig. 46) consists of a closely wound straight helical-filament, incandescent nitrogen lamp of 6 volts, 5 amperes,

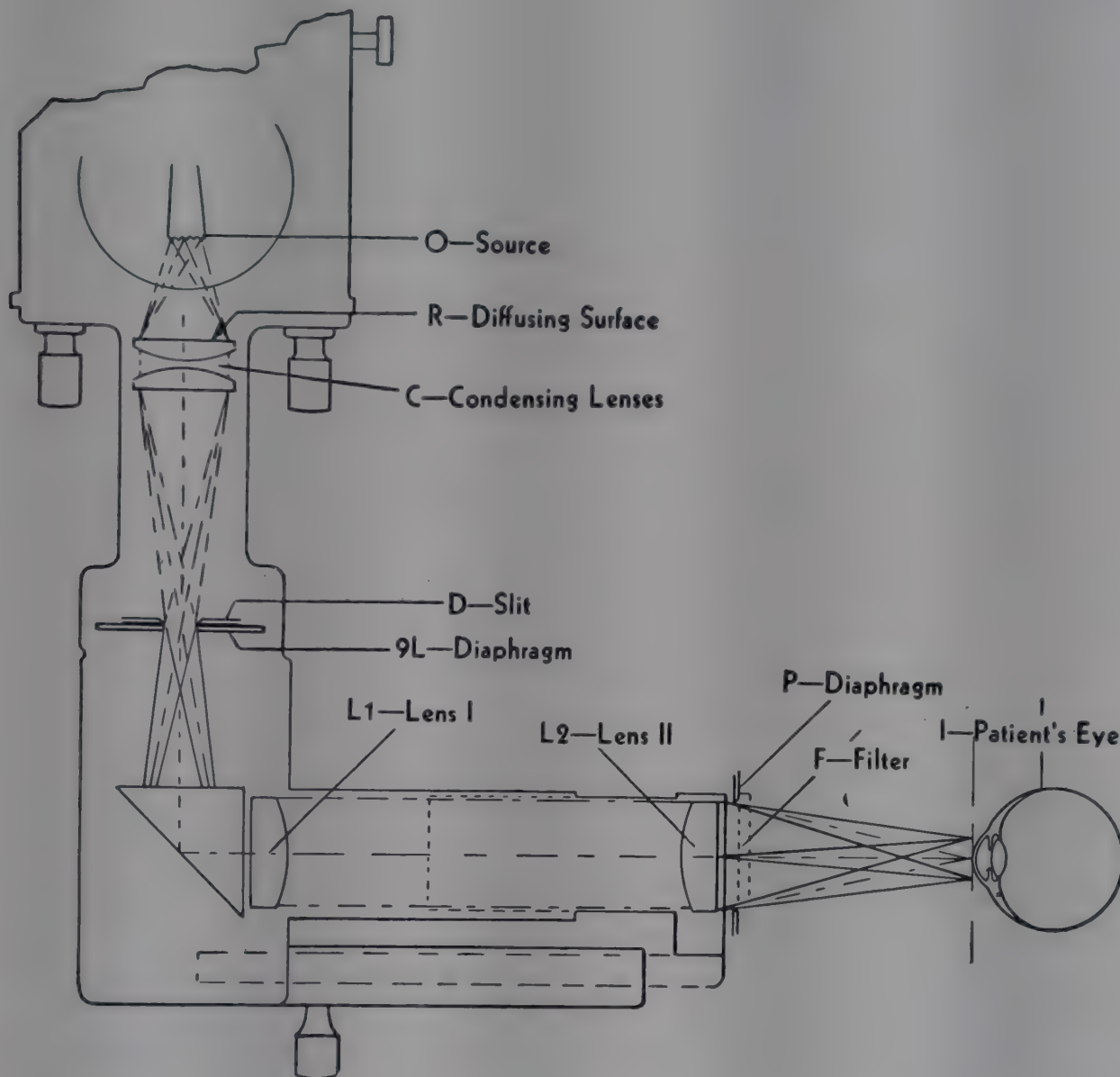


FIG. 46. Diagram of optics and illuminating system of the Bausch & Lomb-Poser model of the slit lamp. (Courtesy of Bausch & Lomb Optical Company.)

to be used with a variable transformer or resistance to furnish light of varying intensities. A specially designed lamp housing is provided to dissipate heat. This incandescent lamp furnishes a light beam of high intensity. The condensing lens system, consisting of two plano-convex lenses with convex surfaces facing each other, the total power of which is approximately 50 D., projects an image of the lamp filament into the aperture of the adjustable slit (Fig. 47). A

fine-grained ground-glass disk in front of the adjustable slit diffuses the image of the spiral filament of the lamp bulb, so that an even illumination of great intensity is obtained. In the Poser slit lamp the collimating lens (L 1) in front of the slit is a doublet of 70 mm. or approximately 14.28 diopters and the objective illuminating lens (L 2) nearest the patient's eye is a diaphragm achromatic doublet (14.28) identical with that in the Koeppe system.*

The adjustable slit is situated in the focal plane of a second condenser lens, which makes the beam of light parallel. This parallel beam of light is then reflected by means of a rectangular prism into the horizontal plane. An aplanatic illuminating lens system, provided with a rack and pinion movement, situated at the end of slit illuminator bar, projects the luminous beam on the portion of the eye to be examined. An individual elevating screw permits vertical adjustments of the beam. With this illuminating system, the light-gathering power is greatly increased as compared with that of the original Gullstrand or Vogt methods. In addition the beam is more sharply defined and free from chromatic aberration.

A revolving disk, situated close to the adjustable slit, is provided with four round apertures, the diameters of which are 1 mm., 3.5 mm., 7 mm., and 8.5 mm. These openings form cylindrical beams of light and also shorten the height of the slit beam.

The milled head of the adjustable slit is situated close to the rotating collar of the elevating device. This facilitates adjustments of the position and width of the slit aperture. Rectangular aperture diaphragms of 6, 10, and 15 mm. width, all 20 mm. in length (marked as No. 200, No. 100, and No. 70 diaphragms) fit in front of the aplanatic illuminating lens system. With the full aperture of the lens or when using the No. 70 diaphragm, the divergence of the rays is so great that the depth in which the slit is in focus is very small but

* The fundamental difference between the Koeppe (also Poser) and Vogt illumination systems is in the position of the filament image with respect to the slit diaphragm. In the Koeppe system there is a light-diffusing glass in front of the bulb to slightly break up the filament structure. This diffused filament image is focused in the slit, which obviously gives a high intrinsic brilliance at the slit. In front of the slit is a doublet condensing lens. This provides parallel light from this lens to the objective condensing lens. Because this light is parallel the distance from the objective lens to the focused slit image is constant.

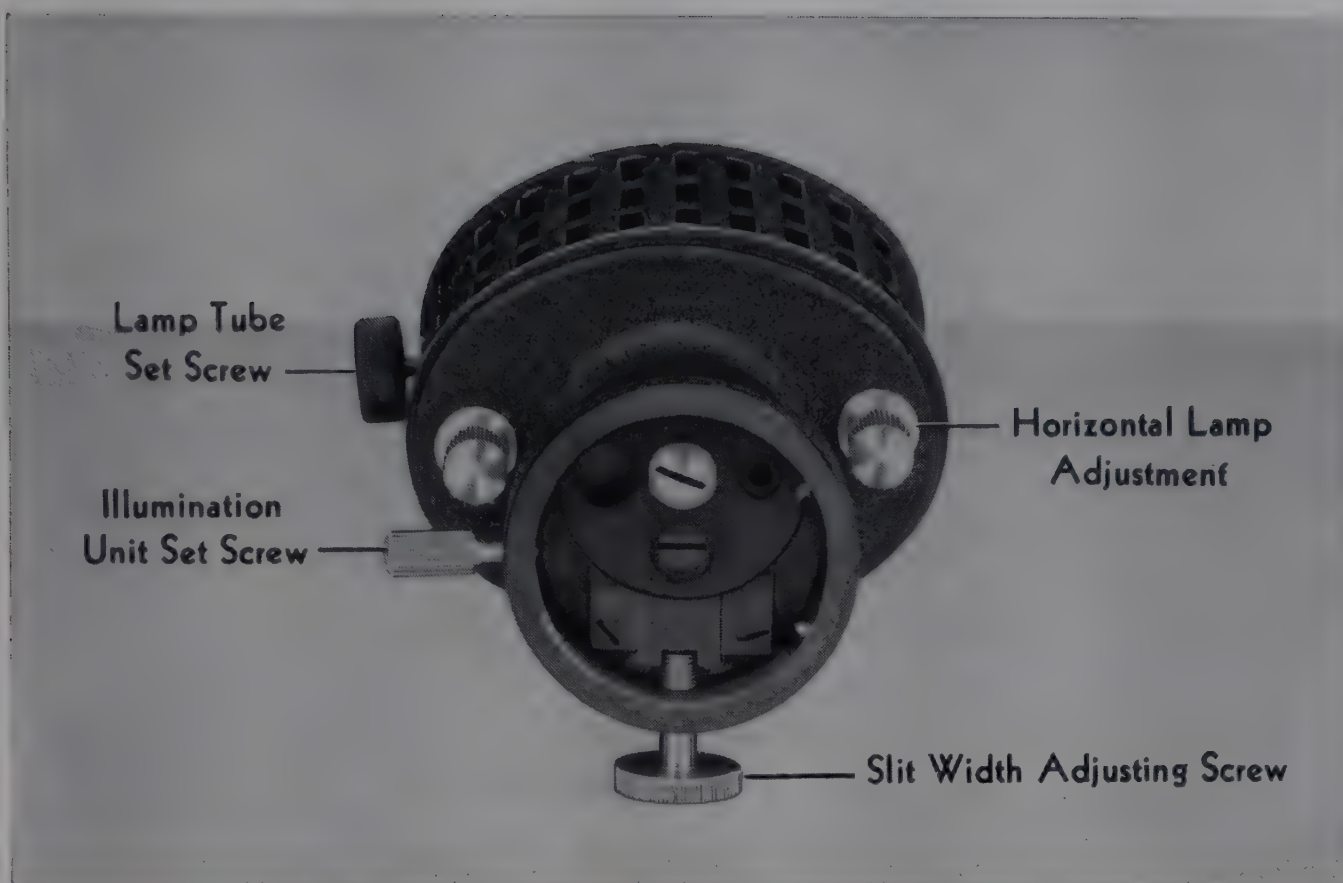


FIG. 47. Illuminating unit of the Poser slit lamp (bottom view). (Courtesy of Bausch & Lomb Optical Company.)



FIG. 48. Diaphragms and filters furnished with the Bausch & Lomb-Poser slit lamp. (Courtesy of Bausch & Lomb Optical Company.)

with the No. 70 diaphragm in position the maximum resolving power is obtained. By using the No. 100 or No. 200 diaphragm, the apparent depth of the focus of the slit is much greater so that a considerable depth of the eye can be examined without refocusing the slit. The effect, as far as the depth of focus is concerned, is the same as though lenses were being used having a focal length in millimeters the same as the diaphragm numbers.

The rectangular aperture diaphragm increases the definition of the illuminated slit image, particularly in focal illumination, which is an important feature in studying the structure of the vitreous and the supporting fibrillar meshwork (Fig. 48).

The $10\times$, 20 mm. aperture (No. 100) diaphragm is recommended for examination of the finest structures. The loss of light with this diaphragm is imperceptible. The use of smaller aperture diaphragms is not advisable, since for the minutest intravital histologic examination the $10\times$, 20 mm. aperture seems to be the limit. The rectangular aperture diaphragms to a large extent prevent scattered light from entering the neighboring field of the illuminated area. The surrounding area of the illuminated field is much darker when the rectangular diaphragms are used.

The diaphragm with the largest aperture has two studs to hold light filters — yellow, green, or blue. Color filters increase definition and make it possible to distinguish color contours of details difficult to recognize with white light. The blue-light filter aids in determining minute differences of the light yellow and dark brown pigment elements of the iris (blue absorbs dark yellow). The yellow-light filter is useful in observing the sclera, conjunctiva, and eyelid margins. Von Hess recommends the yellow-light filter for observers of mature age. The green-light filter is useful in observing the vascular structure and blood corpuscles.

The Koeppe silvered mirror can be attached to the lens mount in the same manner as the diaphragms. The purpose of this mirror is to reduce the angle between the illumination and observation axes so that the deeper portions of the eye can be examined readily. For examination of the deep vitreous and retina, a contact lens with

a plano-anterior surface is required. This form of contact lens* is designed so that it is held in place by the eyelids, resting on the peripheral convex portion of the anterior surface (Fig. 269).

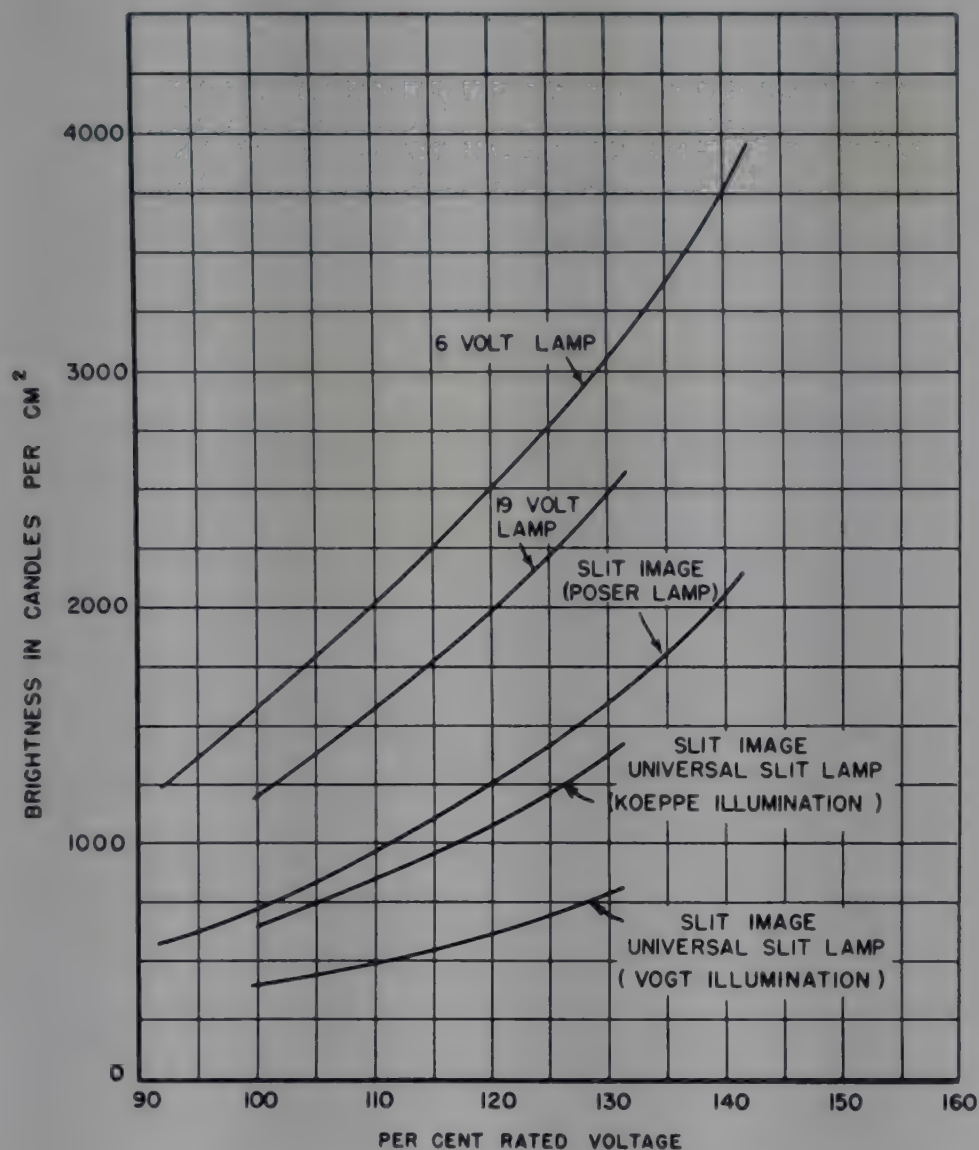


FIG. 49. Brightness of various light sources.

The flexible electric cable of the Poser instrument is passed through the base of the instrument and the slit lamp arm. This is an improvement over the arrangement in the older models in which suspended, exposed cable is so easily jarred and continually interferes with the movements of the observer. The base is fitted with an adjustable transformer and electric switch. The head-rest and the chin-rest are integral parts of the base of the instrument. The chin-rest is fitted with an elevating gear and the head-rest is hinged to permit its inclination. An adjustable screw movement fitted to the head-rest

* Bausch & Lomb fundus contact lens.



brings the eye under observation nearer or farther away from the position of the projected illuminated slit image. This greatly facilitates observation of objects located in the various planes of the transparent media of the eye with a given illumination adjustment.

Brightness of Various Light Sources. In a graph (Fig. 49) showing the comparison of the 19 volt helical filament nitrogen-filled bulb, as used in the Universal slit lamp, and the 6 volt helical filament nitrogen-filled bulb of the Poser slit lamp, it is noted that the Poser bulb is brighter by approximately 425 candles per square centimeter, with both bulbs burning at their rated voltages, indicated by 100 per cent volts at the bottom of the chart.

The focused slit image of the different systems was measured as indicated on the graph. The maximum illumination of the Bausch & Lomb Universal slit lamp, focused in the Vogt manner, is 800 candles per square centimeter, while the Universal slit lamp with the Koeppe illumination gives 1400 candles per square centimeter. The Poser lamp at its maximum voltage gives 2125 candles per square centimeter. Table V indicates the relative differences in brilliance:

TABLE V
RELATIVE DIFFERENCES IN BRILLIANCE OF LIGHT
SOURCES

8-10 volt nitra lamp	(Vogt) 3.3
19 volt nitrogen-filled lamp	(Koeppe) 5.35
6 volt nitrogen-filled lamp used in Poser slit lamp . .	(Koeppe-Poser) 5.9

BINOCULAR MICROSCOPE

The arm, carrying the binocular microscope, is fitted with an elevating gear, similar to that of the illuminating system. The upper end of this gear is provided with a socket into which is fitted the pivot attached to the rack and pinion focusing device of the body tube of the binocular microscope, so that the microscope may be rotated independently and with a clamping screw fixed in any position.

The revolving nosepiece fitted with two pairs of objectives of dif-

ferent powers permits quick changes of magnification. The paired objectives are usually of 55 mm. and 40 mm. focal length, respectively. Huygenian eyepieces of 10 \times are part of the equipment. The eyepieces are of a special wide-field design with a long eye point, in order to enable an observer wearing ophthalmic correcting lenses to use the full field of the microscope. With the previous type of microscope (Czapski) it was necessary for an observer to remove his lenses to use the microscope effectively.

OTHER BIOMICROSCOPES

There are several other types of apparatus available, for example, the Lemoine and Valois,* the Fincham,† and the Mayou,‡ and the Haag-Striet.§ These instruments differ only in certain mechanical improvements or simplifications; optically they offer no new or revolutionary features and consequently have been little used in this country.

In the *Lemoine and Valois biomicroscope* (Figs. 50, 51) the component parts are mounted on a broad aluminum base. The instrument is rigidly constructed in order to avoid vibrations. The illuminating system is suspended from a horizontal arm which is articulated to the main support. A nitra bulb of 150 footcandles is used. Lemoine and Valois state that the instrument is rigid, mobile, and easy to handle.

In the *Fincham apparatus*, the illumination system (Fig. 52) and the microscope are mounted on the arc. The center of curvature of the arc coincides with the focal point of the beam. The lamp is fixed and the microscope moves along the arc. Practically, this means that as soon as the beam is focused on the eye, the microscope is automatically centered toward the illuminated area. By centering the two systems, accurate measurements of apparent optic depth can be obtained. The readings are made by means of a small scale and the Vernier attached to the slide of the microscope.

* Constructed by Gambs of Lyon.

† Made by Clement Clark of London.

‡ Made by Hamblin of London.

§ Made by Haag-Striet of Berne, Switzerland.



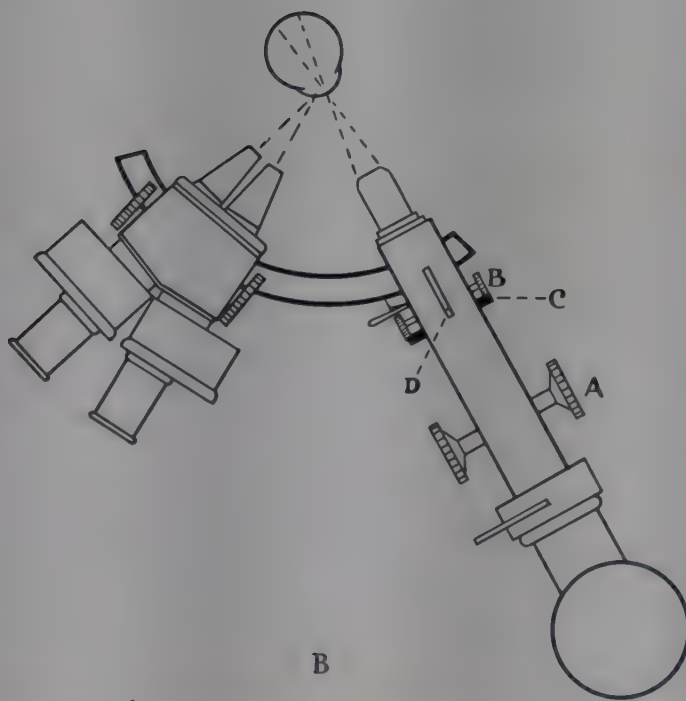
FIG. 50. Biomicroscope of Lemoine and Valois
(early model).



FIG. 51. Later model of Lemoine and Valois biomicroscope.



A



B

FIG. 52. A. Fincham slit lamp. B. Diagram of Fincham slit lamp. (Courtesy of Clement Clarke, Ltd.)

In the *Mayou apparatus*, there is a metal arc (Fig. 53), which is attached to the pillar supporting the microscope. This arc is attached below the microscope, which is in the center of the arc.



FIG. 53. Mayou biomicroscope. (Courtesy of Theodore Hamblin, Ltd.)

Because the illuminating apparatus slides laterally along the arc, the direction of the light is easily and quickly changed.

Haag-Striet (Goldmann) slit lamp. In collaboration with Professor Hans Goldmann, the firm of Haag-Striet, have produced a simplified biomicroscope of excellent design (Fig. 54). In this instrument one supporting spindle carries both the illuminating unit and the microscope so that after the preliminary focusing is done it is possible by means of one single hand control to shift both as a unit. The attachment of a four-sided prism (for double internal reflection) makes it possible to reduce the angle between the illu-

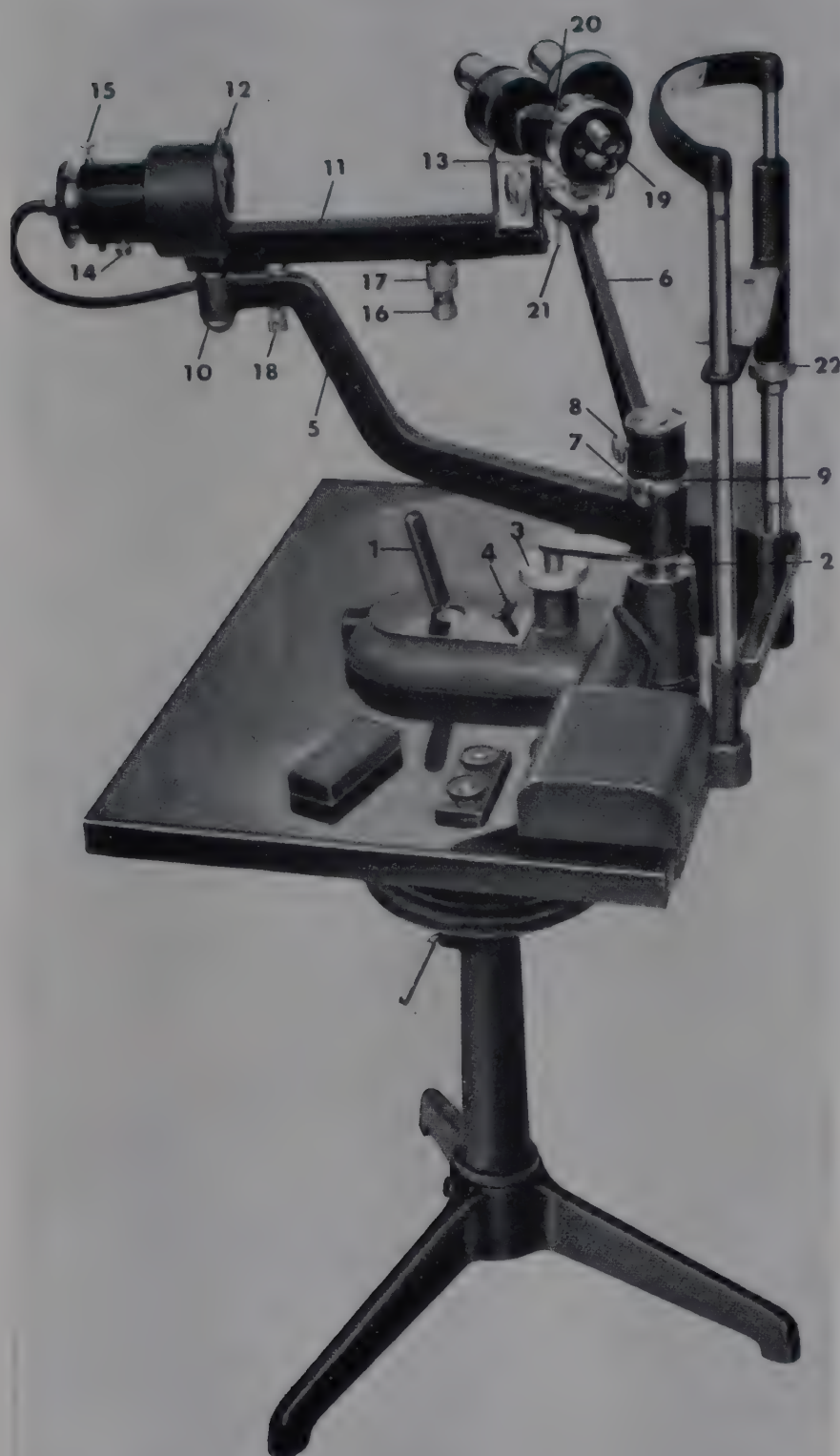


FIG. 54. Haag-Striet (Goldmann) slit lamp. 1, Control lever for affecting both frontal and sagittal movements of the apparatus as a unit. 2, Supporting pillar. 3, Vertical adjustment screw for raising and lowering supporting pillar. 4, Light switch. 5, Lamp arm. 6, Microscope arm. 7, Coupling screw for arms 5 and 6. 8, Locking screw for microscope arm. 9, Scale for reading off angle between lamp arm and microscope barrel. 10, Pivot for lighting equipment. 11, Pivotal arm. 12, Rekoss disk. 13, Condensing lens. 14, Two screws for centering lamp. 15, Locking screw for lamp holder. 16, Knob for adjustment of slit. 17, Focusing knob for condensing lens. 18, Stop for arresting pivotal arm in working position. 19, Objective head, adjusted to low magnification. 20, Lever for altering magnification. 21, Lever arm. 22, Vertical control of chin support.

minating beam and the axis of the microscope sufficiently to permit (with the aid of a contact lens) observation of the fundus background, the posterior portions of the vitreous, and the angle of the anterior chamber.

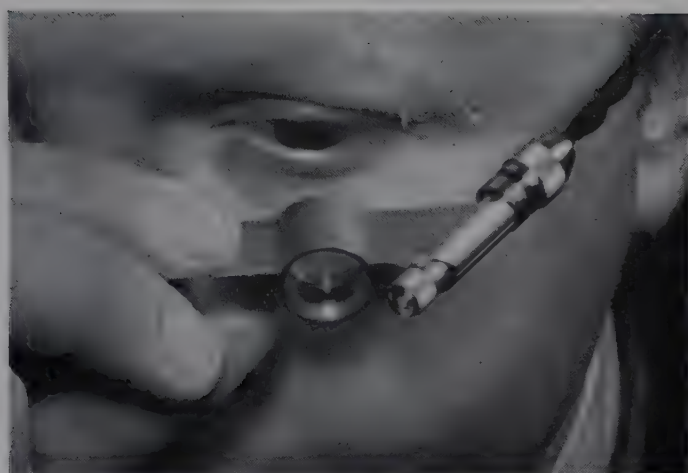
HAND SLIT LAMPS

One great disadvantage of the present-day biomicroscopes is the fact that their size limits their use to the clinic or consulting room. An easily portable apparatus is of value in clinical diagnosis, for example, in determining the presence of aqueous flare, in localization in the cornea and lens, and especially in recognizing massive vitreous obstruction. However, the chief objection to the use of the portable instruments lies in the small size of the slit, the low intensity of illumination, and the low monocular magnification. Focal illumination of this type can be simply obtained by removing the head of a May ophthalmoscope and directing the focused beam into the eye, using a monocular or binocular loupe for magnification. The Wappler U-shaped hair-line filament is peculiarly adapted for this purpose and is a convenient instrument when the vest-pocket battery handle is employed.

The so-called "*featherweight*" *hand slit lamp*, designed by Krimsky,^{180, 181} consists of a 10-power loupe with a channel to hold a lighting unit which permits the ready selection of a vertical slit, a horizontal slit, or a homogeneous circle of light at a fixed focus of 45 degrees to that of the lens. Such selective illumination is obtained not by the use of diaphragms, but by an accurate mechanism which automatically permits the lamp to be brought nearer or farther away from the fixed lens cap in a helical or rotary fashion. The slit beam is comparable to that obtained with the straight filament lamp unit in the hand ophthalmoscope; however, the lamp in this instrument has a square-shaped filament designed by Krimsky, rather than an arch-shaped filament and a pin-hole cap (1/16 inch opening) which fits over the condenser lens cap, thus rendering a truer slit.

To permit free use of both ophthalmoscope and hand slit lamp, the latter unit is operated from a separate medium-sized battery

handle kept in the coat pocket and connected to the slit lamp by means of a fine silk-woven wire. The slit lamp is thus operated by two fingers rather than by two hands; it can easily be slipped into the pocket when not in use.



A



B

FIG. 55. A. Krimsky featherweight slit lamp. B. Krimsky featherweight slit lamp with eyeglass slip-over attachment. (Courtesy of Dr. E. Krimsky.)

An eyeglass slip-over attachment is also provided; this has a channel to permit insertion of a common light carrier, as for the hand slit lamp. This attachment has a longer focus lens of 4-power magnification, which is less satisfactory for diagnostic requirements but suitable for minor surgical procedures, such as removal of foreign bodies, or sutures, or epilation of eyelashes. The slit lamp makes it easily possible to bring foreign bodies into proper relief, and the fixed attachment of the unit to an eyeglass enables the surgeon better to control the patient and to obtain fixation of the eyelids with free use of both hands (Fig. 55).

The *Bausch & Lomb hand slit lamp* consists of a combined magnifying and illuminating system mounted on a battery or cord handle. The observation system consists of a triple aplanatic focusable

ocular magnifier of $7.5 \times$ power. It may be adjusted so that its focal point coincides with the focal point of the luminous beam and may be fixed in this position. The magnifier is so mounted that the



FIG. 56



FIG. 57

FIG. 56. Bausch & Lomb hand slit lamp. (Courtesy of Bausch & Lomb Optical Company.)

FIG. 57. Bausch & Lomb hand slit lamp in use. (Courtesy of Bausch & Lomb Optical Company.)

angle between the illuminating beam and the visual line is small enough to permit study of the posterior lens capsule and anterior vitreous through a pupil as small as 4 mm. in diameter. The magnifier is attached to the barrel of the illuminating system in such a way that it can be rotated from one side to the other in order to view the eye from each temporal side.

The illuminating system incorporates the customary coiled filament ophthalmoscope bulb, a concave reflector situated behind the lamp-bulb, a Rekoss diaphragm disk with three circular apertures and two consecutive slit apertures one of which is vertical and the other horizontal. The lens system projects a brightly illuminated image of the diaphragm aperture into the observed eye. The tube containing the entire illuminating system is mounted horizontally with respect to the vertical handle (Figs. 56, 57, 58).

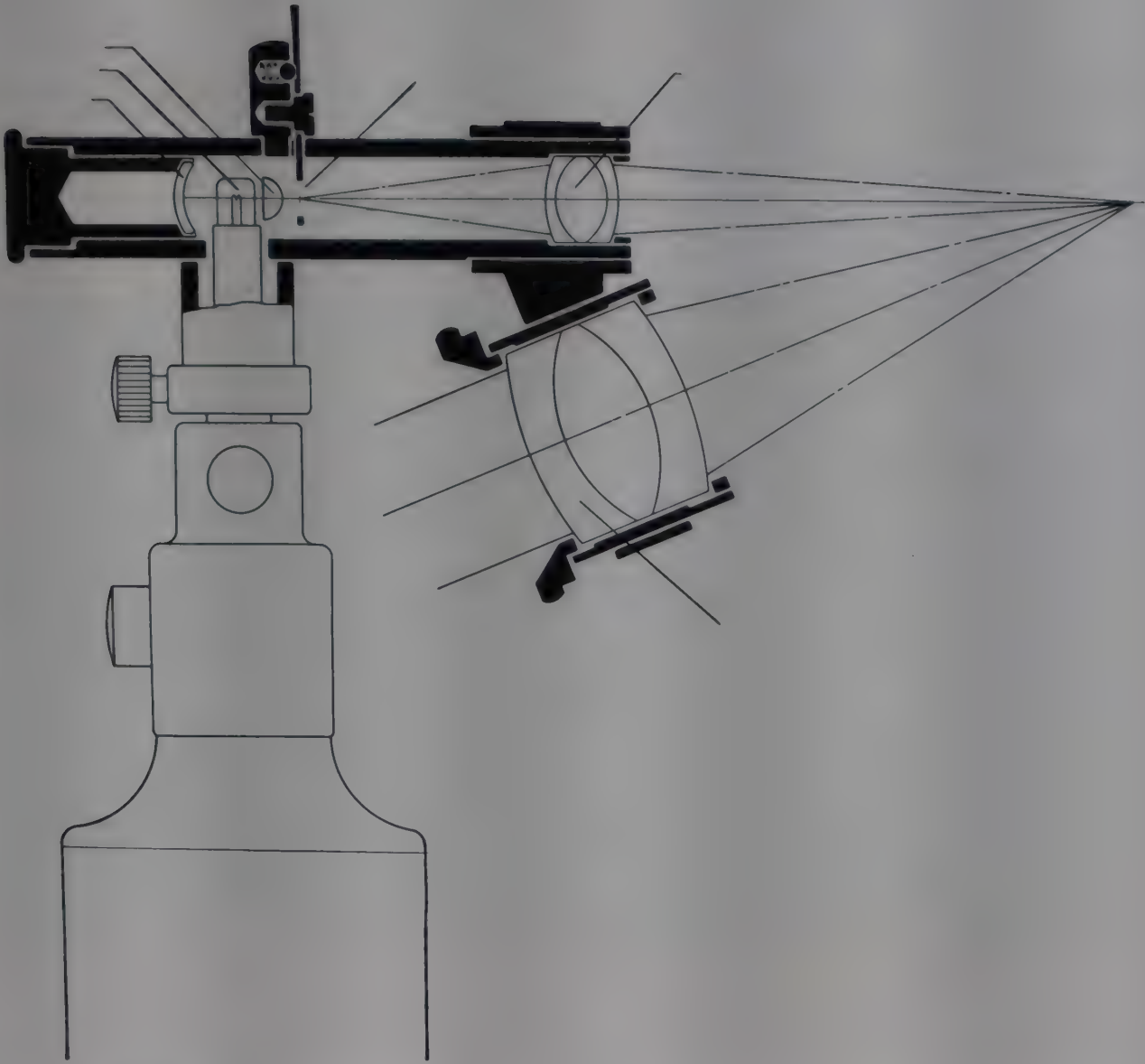


FIG. 58. Diagram of optics of the Bausch & Lomb hand slit lamp. (Courtesy of Bausch & Lomb Optical Company.)

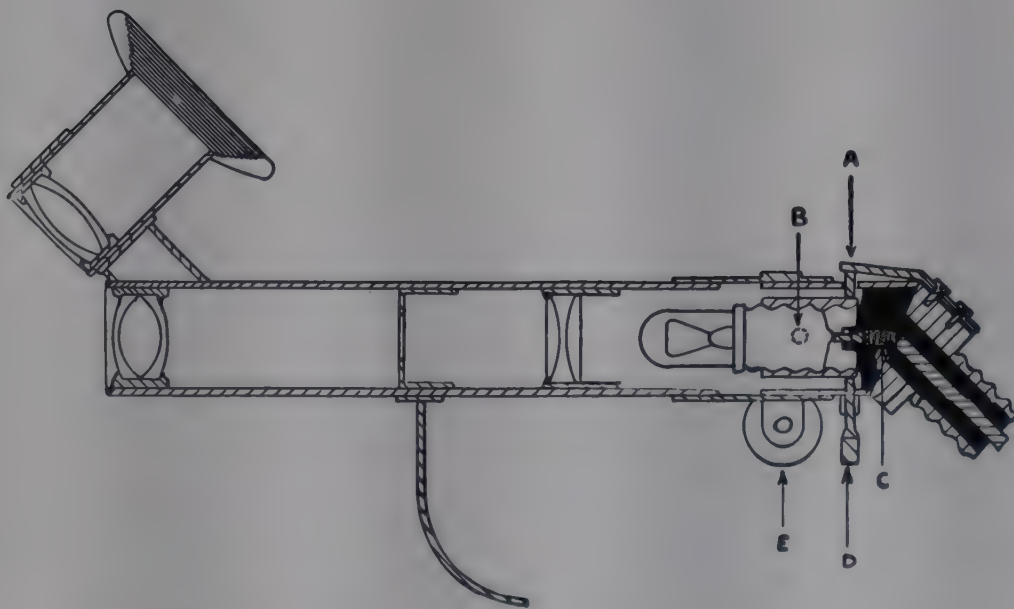


FIG. 59. Diagram of Bishop-Harman hand slit lamp. (Courtesy of N. Bishop-Harman.)

*Bishop-Harman*³¹ designed a hand slit lamp which embodied certain added features. The ocular magnifier (a triple achromat), giving a linear magnification of $12\times$, is replaceable by others of higher



FIG. 60. Bettman and McNair slit lamp is used as a hand instrument or mounted on a stand.

power. The illuminating system (attached to the eyepiece at an angle of 45 degrees) includes a gas-filled, single-strand filament lamp and a powerful condensing lens system, producing an image of the filament on a slit fixed within the tube (Fig. 59). A projection lens (60 D. achromat) at the end of the tube acts as an illuminating lens, producing a clear image of the slit free from disturbing haze or filament irregularities. The lamp is fitted to a battery handle at an angle of 45 degrees.

A portable slit lamp has recently been designed by *J. Bettman* and *G. McNair*. The instrument may be held in the hand by means of a pistol grip or it may be supported on a small stand (Fig. 60). In either case, a head rest steadies it by firm pressure against the patient's forehead, the patient's head being braced against the back of a chair or against the pillow if he is in bed. Magnification is obtained by an ordinary monocular microscope with an erecting system. A $10\times$ ocular and 48 mm. objective are used. Magnification may be varied from 8 to 25 diameters by altering the length of the

tube. The microscope is focused on a rack and pinion with a wheel on either side of the instrument. The illuminating and condensing systems are supported by an arm attached to the grip and move independently of the microscope. The lamp which operates on a 7 volt transformer with a 3-ampere output has a special rectangular-shaped filament. The condenser focuses the filament in the slit, the image of which is projected on the illuminating lens by means of a collimating lens. The width of the slit can be varied at will. The length can be altered from a bundle of light 0.5 mm. in diameter to a slit 5.5 mm. long.

The designers particularly stress the fact that the instrument is portable and less expensive than the usual types.

Chapter Three

TECHNIQUE OF BIOMICROSCOPY

IT is frequently stated that the technique of biomicroscopy requires special skill and is difficult to master. However, in my opinion, anyone who has had some experience with the ordinary laboratory microscope and who understands the fundamental principles of optics can develop a high degree of proficiency in biomicroscopy.

PRELIMINARY SURVEY OF EYEBALL

When a patient is first seen, it is most important to make a thorough preliminary survey of the eyeball by means of ordinary methods, that is, daylight inspection, diffuse and oblique illumination, and ophthalmoscopy. With a knowledge of the macroscopic appearance of the eye, the student is less likely to become confused or disoriented by the myriad of details which appear microscopically. A preliminary survey also tends to shorten the time of examination, which must be brief when a highly inflamed, photophobic eye is to be examined. In these cases, it may be helpful to have an assistant who can steady the patient's head and assist, if necessary, in separating or everting the eyelids (for examining the upper tarsal conjunctiva).

ILLUMINATION OF ROOM

The examination is best made in a semidarkened room in which there is just enough light to permit the examiner to move about and to guide the patient. However, there are occasions, as when one uses low intensities of illumination, when it is expedient to work in complete darkness.

The examiner's eyes should be partially dark-adapted to ensure sensitivity to low intensities of light, such as are obtained with narrow slit beams. Quiet, good ventilation, and proper seats of adjustable height aid in securing the patient's co-operation during long examinations.

Luminous fixation targets (radium paint) on the wall facing the patient will help him to follow directions for moving his eyes; for example, consecutive numerals will give the cardinal directions of gaze.

PREPARATION AND MANAGEMENT OF THE PATIENT

The patient must be as comfortable as possible, if restlessness and fatigue are to be avoided. He should be seated on a revolving backless stool, adjusted to suit his height. The table, chin-rest and head-guard should be raised or lowered so that when the patient's head is in position, he can rest his arms on the table and be completely at ease. The patient should be asked to keep his forehead pressed against the head-guard in order to ensure immobility.

In addition, the microscopist is urged to bear in mind the following points: (1) All drops, especially anesthetics, instilled into the eye tend to produce disturbances of the corneal epithelium, which may be mistaken for pathologic change. (2) Ointments or oils form a greasy film over the conjunctiva and cornea. (3) Examination with the wide luminous beam should be made as brief as possible because of discomfort to the patient. (4) Unnecessary exposure of the retina to the light of the beam should be avoided. (5) A clean chin-cup or celluwipe cover is reassuring psychologically.

ADJUSTMENT OF THE INSTRUMENT

Before making an examination, it is important to ascertain whether the light is focused properly. When using the older apparatus, with Vogt's method of illumination, a small white card, for example, a visiting card, should be placed in front of the illuminating lens to determine whether the image of the filament is properly centered and focused. With the newer apparatus it is necessary only

to have the maximum intensity of uniform light fall directly on the surface of the illuminating lens.

Before actual microscopic observations are made, the microscope and the lamp should be adjusted to the approximate angle and focus desired. The slit beam should be directed either on the closed upper eyelid or on the sclerolimbic region in order to minimize exposure to light. Needless to say, the eyepieces and objectives should be properly and fully inserted in their slots. If the microscope is not fitted to a mechanical stage, the glass plate on which its base slides should be kept cleanly polished in order to facilitate easy movement. As a rule, it is better to have the light on the temporal side.

When the adjustments are completed, the patient should open his eyes and fix his gaze in the desired position (never toward the light). Some microscopists believe that a low voltage, dimly glowing lamp is a valuable aid in obtaining steady fixation. Others simply instruct the patient to look at the microscope. The angle between the illuminating beam and the axis of the microscope should be approximately 40 degrees. However, this angle varies greatly according to the method of illumination employed and the depth of the tissue to be observed. Although the axis of the microscope can be inclined forward and backward, because of the difficulty in focusing with these angles, it is preferable to have the patient shift his gaze (Fig. 61).

At times it is important to have a narrow angle between the axis of illumination and observation. This is especially true in studying the angle of the anterior chamber, the vitreous humor, and the fundus. To obtain a narrow angle a Kleefeld silver mirror is attached to the illuminating lens. When this is done, the microscope and the direction of the arm of the illuminating system are apparently at right angles to one another. The silver mirror is above the median line of observation of the microscope and slightly inclined. The binocular tube of the microscope is tilted upward an increasing amount (Fig. 62). In this way it is possible to obtain a sufficiently acute angle between the axis of illumination and ob-

ervation so that the latter almost corresponds to the direction of the illuminating rays.

From this point on, the observer focuses the microscope with one

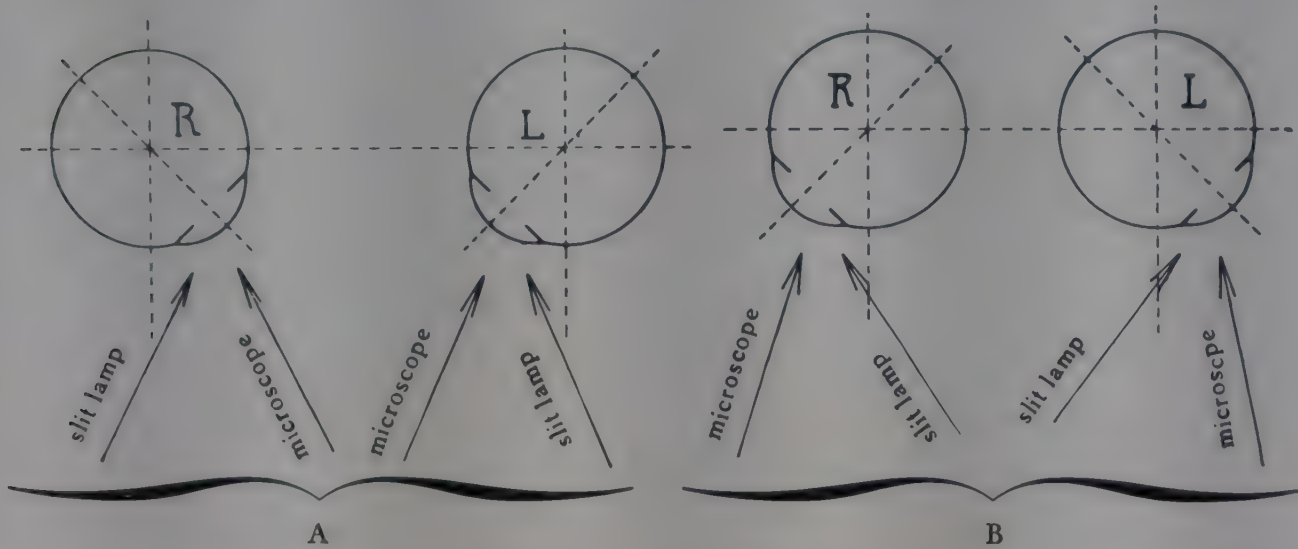


FIG. 61. Diagram showing positions of the eye, slit lamp, and microscope for best examination of the peripheral parts of the cornea in order to avoid distortion of the parallelepiped. A. Illumination from the temporal side. In examining the right eye the surgeon should operate the microscope with the right hand and the slit lamp with the left hand and vice versa for the left eye. B. Illumination from the nasal side (over the bridge of the nose). In examining the right eye the microscope should be operated with the left hand and the slit lamp with the right and vice versa for the left eye. (Courtesy of Bausch & Lomb Optical Company.)

hand and the illuminating lens with the other, resting his elbows on the table. He is thus able to follow the movements of the patient's eye and to make rapid compensatory focal adjustments either of

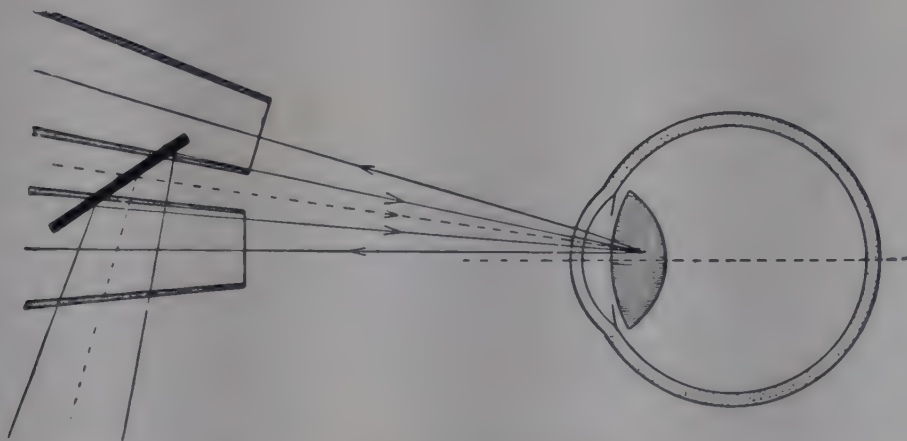


FIG. 62. Adjustment for observation of the lens using Kleeefeld's silvered mirror. (Courtesy of Carl Zeiss, Inc.)

the beam or the microscope (Fig. 63). Practice in co-ordinating these two focusing movements greatly increases the speed and ease of examination. The student is advised to employ low magnifications,

not only because of the large field and depth of focus but also because of the ease of orientation.



FIG. 63. Making an examination with the Bausch & Lomb-Poser slit lamp.
(Courtesy of Bausch & Lomb Optical Company.)

STEREOPSIS

The biomicroscope is so constructed that it affords stereoscopic vision. Some of the principal accomplishments obtained by biomicroscopy are the result of this factor. If the stereoscopic vision of the observer is impaired by anisometropia, astigmatism, or muscular imbalance, these defects should be corrected by glasses. The pupillary distance of the oculars should be carefully adjusted to correspond to the interpupillary distance of the observer.

How the mind forms a concept of solidity from a flat retinal image is still a moot question. The modern theory is that the brain interprets actual visual sensations in the light of judgments derived from past experience.* Perception of depth is produced by static

* A thorough study of the part played by the synthesizing power of the brain will not be discussed here. Attention will be confined strictly to ocular phenomena.

and dynamic factors. The static factor depends on the slight disparity between images presented to the two eyes. This difference between the two images is caused by static parallax. The dynamic factor depends upon the parallax displacement of objects relative to each other when the eyes are moved.

According to von Helmholtz (1866), perception of solidity by one-eyed persons is achieved by variations in the retinal images resulting from constant involuntary movements which are substituted for the normal binocular act.

Since the binocular microscope used in biomicroscopy permits stereoscopic visualization, the static factor, which with dynamic factors produces depth perception, will not be troublesome. However, the small involuntary movements which afford normal parallax are hampered by the use of the microscope. When studying an object by binocular vision, the combined voluntary and involuntary eye movements result in a form of "visual palpation." In biomicroscopy of the eye "visual palpation" can be simulated in two ways: (1) by altering the perspective, which is completed by changes in observation and illumination; and (2) by varying the type of illumination.*

METHODS OF ILLUMINATION

Variations in the manner of projection of the beam and in the intrinsic optical properties of the eye afford the examiner six techniques of illumination: (1) diffuse illumination; (2) sclerotic scatter; (3) direct focal illumination; (4) examination by retro-illumination; (5) examination in the zones of specular reflection; (6) indirect illumination.

DIFFUSE ILLUMINATION

This type of illumination is especially useful in studying the topography of pathologic changes. It can be obtained either by throwing the beam out of focus or by interposing a ground glass.

* A further extension of the method of visual palpation has been suggested by Koepe. He advises imparting an oscillatory motion to the beam; this produces the dynamic factor mentioned above, by causing apparent parallax displacement of objects with relation to each other.

In other words, a beam of light, composed of either converging or diverging rays, is used. With the Comberg instrument, in which a ground glass can be slid into position in front of the illuminating lens, it is possible to obtain a circular spot of strong diffuse light, which covers the entire orbital region. The type of illumination thus obtained is similar to but more intense and uniform than ordinary oblique illumination with the condensing lens. In addition, stereoscopic magnification is obtained by the binocular microscope.

The advantage of this type of illumination lies in the fact that the entire surface of the cornea, iris, or lens may be viewed. For example, the entire extent of a fold in Descemet's membrane, a corneal nerve or scar, or the whole configuration of the lens capsule or surface of the adult nucleus may be seen at a glance. However, this method does not provide the advantages afforded by direct focal illumination with the focused slit beam.

SCLEROTIC SCATTER (S. S.)

This important method of illumination, described by Vogt and later employed by Graves, depends on the dispersion or scattering of light, which is evident in a translucent tissue. The focused beam is directed on the corneoscleral limbus. The marked dispersion and scattering of light which occurs in the perilimbal sclera, produces a crescentic halo of light around the cornea, particularly marked on the opposite side. Moreover, after internal reflection, the light passes through the cornea (Fig. 64).

If the cornea is normal, the light passes through it unimpeded and unobserved, except for a narrow faint haze at the limbus owing to the relucency of the superficial limbal spur. If, however, there is a disturbance of the normal transparency, e.g., a nebula, macula, keratocele, interstitial deposit or perforating scar, this is made visible by the light-scattering properties of the pathologic tissue.

An analogy to this method of illumination is seen in the transmission of light internally by an angulated rod of lucite. By internal reflection it carries the light around the bend and delivers it to the roughened translucent extremity from which the light is diffused;

for example, in a modern type of lucite surgical retractor.

In the ideal method of examination one should employ sclerotic scatter early to detect any changes in corneal relucency. This has

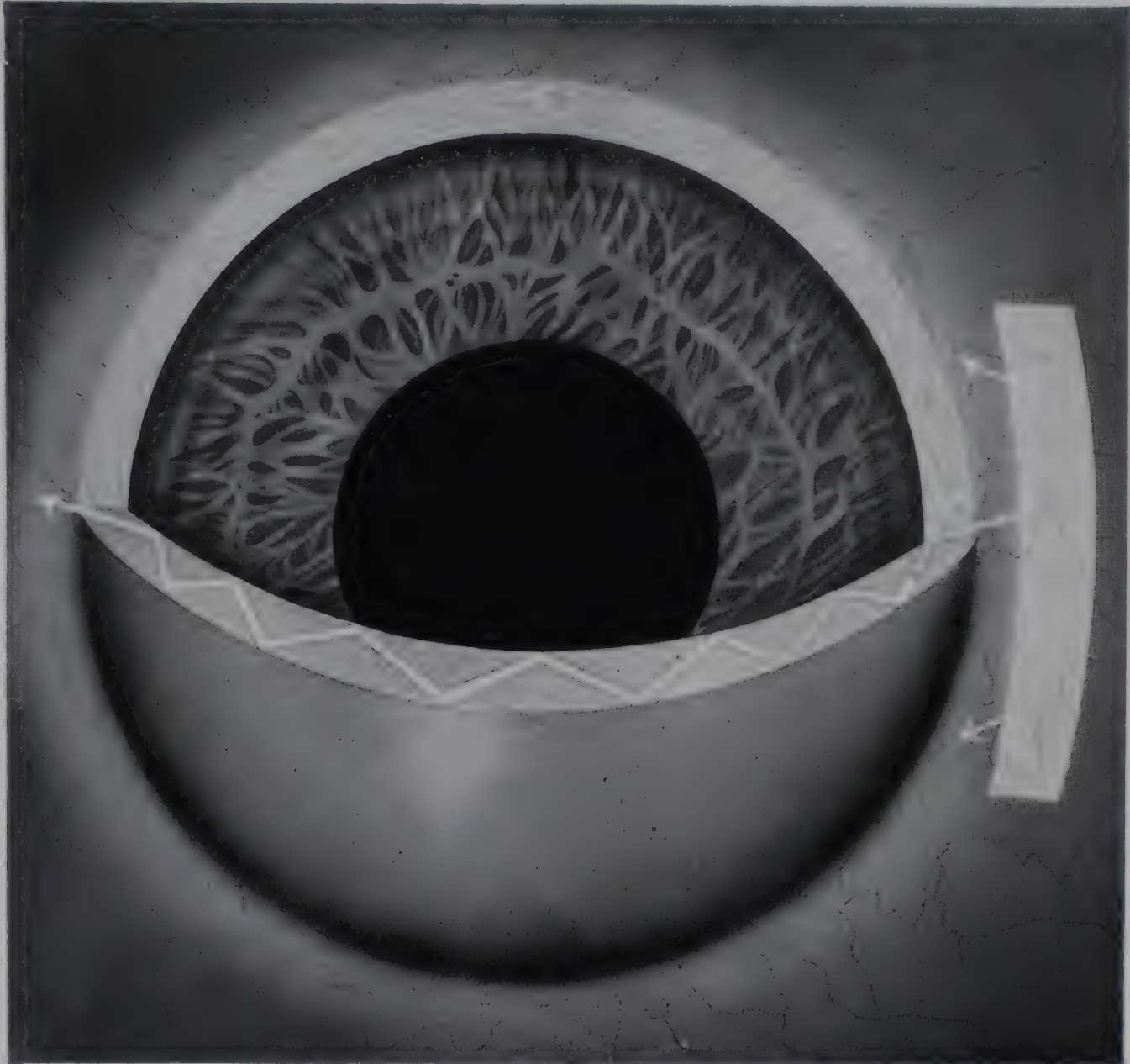


FIG. 64. Sclerotic scatter. Diagram (upper part of the cornea cut away) showing pathway of light as it traverses cornea by total internal reflection; halo of light is particularly marked at the opposite corneoscleral junction; the scar in the center of the cornea is caused to stand out by obstruction and scattering of the internally reflected light. (This is a form of darkfield illumination.)

the merit of gradually accustoming the patient to the light without internal glare. It also saves time in recognizing even the faintest pathologic change (nebula, vacuole, deposit, blood vessel, or other feature), which only moderately obstructs light, but the responsive power of which permits observation under illumination by sclerotic scatter (Fig. 65).



FIG. 65. Superficial corneal opacities by sclerotic scatter.

DIRECT FOCAL ILLUMINATION (D. F. I.)

The use of the focal beam in examining ocular tissues constitutes the basis of all methods used in biomicroscopy, which consist merely in an elaboration of this basic technique. In using direct focal illumination (Fig. 66) the focal point of the beam is regulated until it coincides with the exact focus of the microscope. To ascertain whether the focal part of the beam is being used, the following test may be made: Intercept the light from the illuminating lens with a small card and determine the point at which the illuminated area becomes smallest and most intense. This point is usually at 7.5 cm. from the usual illumination lens (7 cm. focal length).

As the focused part of the beam * strikes the eye, the optical ef-

* Unless otherwise stated, when referring to the beam, it should be understood that the slit opening is used and that its shape is rectangular.

fects produced depend on the degree of transparency of the tissues it traverses. For example, if the beam is focused on a relatively non-transparent tissue, like the sclera or the iris, a brilliant and sharply



FIG. 66. Diagram of direct focal illumination (dissected view, upper half of cornea has been removed). The formation of the corneal and lens blocks by the flux of the illumination (focused beam) is shown.

defined area of surface illumination is obtained, most of the light being reflected, scattered, or absorbed. But if the beam passes through transparent or relucant media, like the cornea or lens, an opalescent block of light is formed. Although the cornea seems entirely transparent in diffuse light, the focused beam reveals that this is not absolutely true. Actually, there are no completely transparent media in nature. Complete transparency could only occur in a vacuum. Media like the cornea and lens have a complex cellular structure and are consequently heterogeneous in type, in contrast to a uni-

formly homogeneous medium, such as glass. The internal dispersion of light caused by the heterogeneity of these semi-transparent tissues is known as *relucency* (Graves).

Loss of intensity of light results when a luminous beam traverses an optic medium. This loss is caused by reflection, refraction, dispersion, and the like. The loss of intensity when the beam passes through a medium, reveals the optical density of the medium by the degree of relucency present. In the eye, these phenomena account for the formation of opalescent blocks. Because the beam is made to traverse the cornea obliquely, the shape of the resulting rectangular block is a parallelepiped prism, the external and internal surfaces of which are slightly curved owing to the anatomic shape of the cornea; the degree of curvature depends on the angle between the corneal axis and the illuminating beam. A corneal scar or infiltration increases the localized optical density. Consequently, when viewed in direct focal illumination, a corneal scar or infiltration appears whiter than the normal opalescence of the corneal block. Fluid in the lens cortex in the form of a water-cleft (as in incipient cortical cataract) appears black in focal illumination in contrast to the surrounding normal opalescence of the lens. These dark fluid spaces are merely areas of lesser optical density.

The Parallelepiped. The parallelepiped has been given various names: "prisma" in German, "prisme rectangulaire" in French, and "truncated prism" or "optical block" in English. In the cornea this parallelepiped figure or block may be compared to an ice cube. Since the cornea is a divergent meniscus, the vertical edges, although curved, are not exactly parallel. The anterior surface of the parallelepiped is called the anterior band or anterior face and the posterior is called the posterior band or face. These faces correspond to the anterior and posterior surfaces of the cornea. In Figures 67 and 68, *abcd* represent the anterior surface of the cornea and *efgb* the posterior corneal surface. The two walls *acge* and *bdfh* are actual surfaces; they limit the width of the beam and represent the internal layers of the cornea. The proximal side, *acge* is more sharply outlined than the distal side, *bdfh*, which at times may be poorly dis-

cernible. The edge *ac* is important because it distinctly marks the boundary between the corneal surface and the internal layers of the cornea. This feature can be demonstrated by the instillation of a 3



FIG. 67

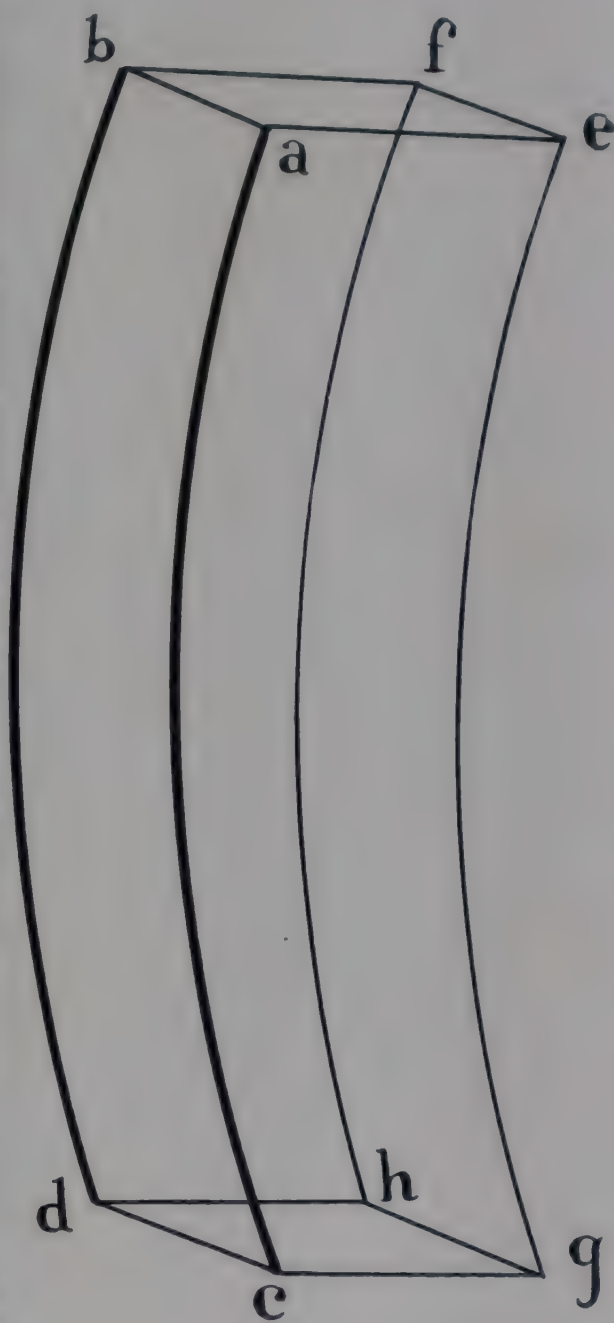


FIG. 68

FIG. 67. Corneal parallelepiped. *abcd*, Anterior surface (or face) of cornea; *efgh*, posterior surface (or face) of cornea. The edge, *ac*, represents the line at which the light begins to penetrate the cornea. (The light comes from the right.)

FIG. 68. Diagram of parallelepiped. *abcd*, Anterior surface of the cornea; *efgh*, posterior surface of the cornea. (The light comes from the left.)

per cent solution of sodium fluorescein, which stains the tear film surface of the anterior face green. Within the normal corneal block delicate branching nerve fibers are observed. Similarly, when the beam is directed into the pupillary area and allowed to pass through

the lens, an opalescent block or wedge is formed. The dark area between the corneal parallelepiped and the lens block represents the depth of the anterior chamber. The lens block (Fig. 6) is composed



FIG. 69. Optic section of the cornea. As the beam is narrowed by narrowing the slit the lateral walls of the parallelepiped approach each other, producing a virtual plane or section. The first of the lines on the surface of the cornea is the precorneal tear film line; the dark space between this and the succeeding light line represents the nonrespective epithelium; behind this is the more reluctant Bowman's zone; then follows the parenchymal thickness of cornea, the deep edge of which (*efgh*) represents the posterior corneal surface. *abcd*, Anterior surface of the cornea.

of several successive bands of light (Fig. 70). These bands are produced by reflections from the surface of the zones of discontinuity and represent the optical delineation of the internal lens architecture. Since the sagittal diameter of the lens is from four to five times that of the cornea, its entire thickness cannot be observed with one focus

of the microscope. The same is true of the beam; when the convergent "focused part" of the beam is situated in the anterior part of the lens, the deeper or posterior parts are viewed in diverging

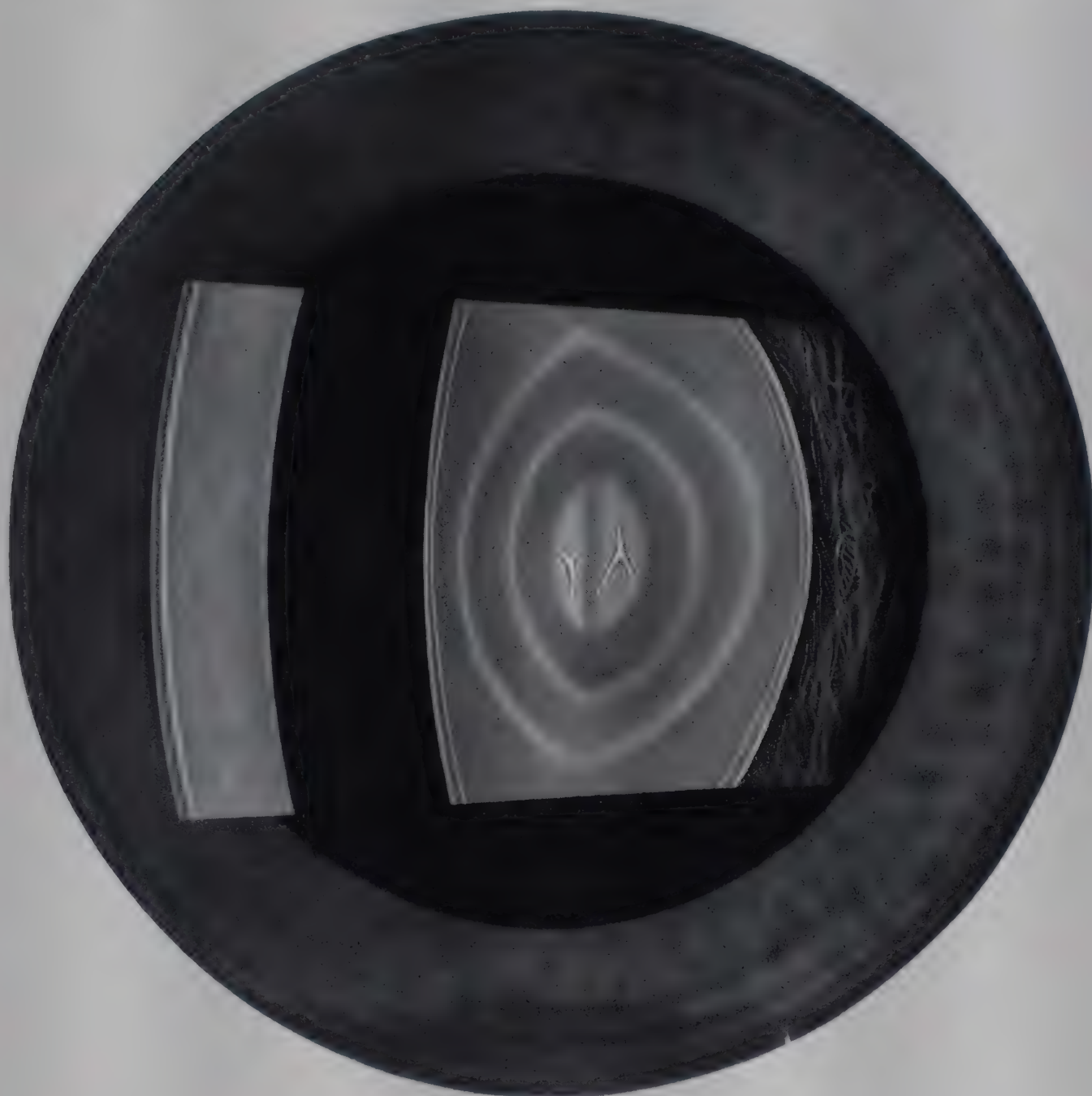


FIG. 70. Optic sections in the cornea and lens produced by the narrow beam. The section in the lens is a composite view because when the anterior part of the lens is in focus the posterior part is viewed by diverging rays and hence is not in sharp focus. (Semidiagrammatic.)

(postfocal) rays; consequently, the beam must be refocused deeper in order to reveal details in this area. Thus, in examining the lens, both the beam and the microscope must be successively refocused on its anterior and posterior portions in order to obtain a composite view.*

* By using a diaphragm (Poser slit lamp, Bausch & Lomb Optical Company) an apparent increase in depth of focus of the beam can be obtained and consequently less refocusing is required in studying the lens (page 48).

Beyond the lens block the gossamer-like vitreous structure is seen. The illuminated portion of the vitreous is comparatively dim and its outlines are poorly defined. It will be remembered that almost 85 per cent of the luminous intensity of the beam is dissipated as it passes through the cornea and lens. Because of this loss of illumination and also because the angle between observation and illumination cannot be narrowed beyond a certain degree, only the anterior third of the vitreous can be examined.*

The Optic Section. The dimensions of the light beam are directly proportional to those of the screw-operated slit (Fig. 107). As the slit is opened, the width of the optical block increases and, consequently, the widths of the anterior and posterior surfaces are wider. This interferes to some extent with good observation and localization in the layers between them, that is, the corneal stroma and the middle layers of the lens. By narrowing the slit and the beam, the widths of the anterior and posterior faces of the optical block are reduced. But the anteroposterior (sagittal) depth of the block remains unchanged. By continuing to narrow the light beam by diminishing the slit to 0.5 mm. or less, the width of the anterior and posterior faces eventually corresponds to a line about 20μ thick (Figs. 69, 70). In this way a veritable optic section of the tissue is obtained. This affords an ideal means of studying the tissue between the surfaces of the optic section and of precisely localizing structures in this zone. Naturally, as the beam is narrowed, its intensity is decreased but because of lesser dispersion and reflection of light in the tissues, this affords a greater contrast between light and dark, resulting in the increased visibility of minutiae. To obtain the full benefit of this process the eyes of the observer should be dark-adapted. Students often fail to realize that the optic section represents a sagittal or coronal view and not a frontal view. It should be remembered that the narrow beam is like a knife which cuts the tissue through its entire thickness, exposing its internal features.

This property of the narrow beam (i.e., permitting visualization

* However, the first obstacle may be partially overcome by the use of the arc lamp, and the second by special devices, such as monobjective microscope, Koepe silvered mirror, and corneal contact glass.

and localization by means of optic sections, analogous to the serial sections in histology) is of the greatest importance. As in tissue studies, the thinner the section cut by the microtome the better the microscopic characteristics, so in biomicroscopy, a thin optic section affords most accurate information. The failure of students to employ the narrow beam is the greatest single obstacle to securing the finer nuances in biomicroscopy.

RETRO-ILLUMINATION

This method was originally termed transillumination but in order to avoid confusion with internal trans-scleral illumination (diaphanoscopy), Graves suggested the name retro-illumination (Figs. 71, 72). *Examination by means of retro-illumination may be defined as the observation of normal or pathologic structures (in transparent or semitransparent media) in light reflected from tissues situated more posteriorly* (Fig. 73). The cornea and the lens offer the best field of application for this method. When the beam is directed obliquely through the cornea, some of the intensity of the light is consumed in forming the block. A greater portion, however, enters the interior of the eyeball; if this light falls on the nontransparent iris, most of it is reflected back through the cornea. Therefore, if the observer looks to one side of the corneal block (in the direction of the illuminated iris) a diffuse yellowish area of shadowy illumination is observed on the cornea; the color of this light depends on the color of the iris. The pupillary area can be used as a reflecting screen if an opaque cataractous lens or a dense pupillary membrane is present. The whitish light from the zone of specular reflection from the posterior lens capsule permits retro-illumination of the anterior portions of the lens, the iris border, and the cornea. The use of the cylindrical bundle, as obtained with the stenopeic pinhole in the aperture disk, is recommended when employing this source of reflected light.

It is well to realize that the same objects may present one color in direct illumination and another in retro-illumination. For example, a capillary seen in the corneal block appears white, whereas

by retro-illumination, it is reddish yellow. Moreover, the color of such features as droplets, the index of refraction of which is less than that of the surrounding media, depends on the color of the re-

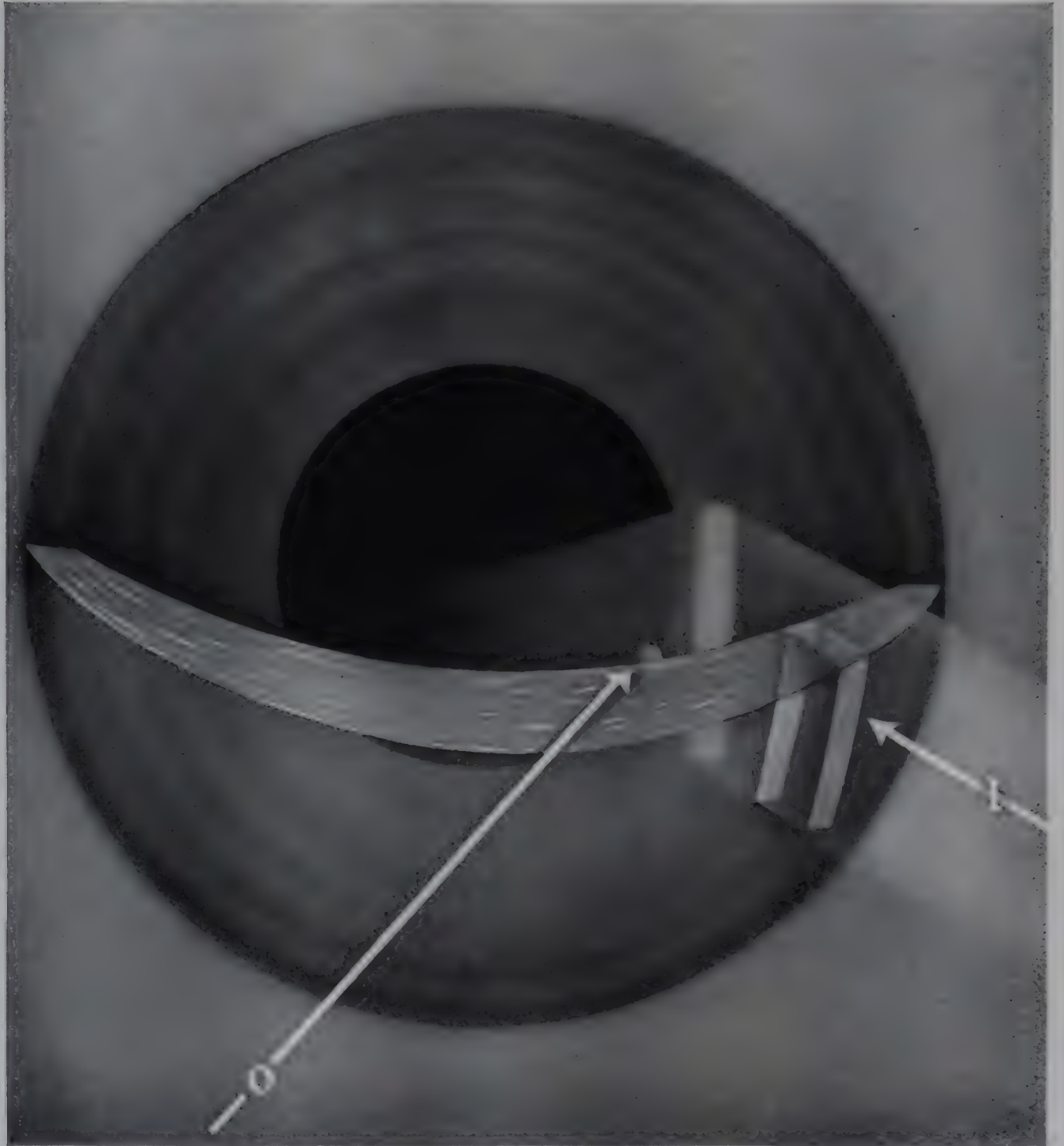


FIG. 71. Diagram of direct retro-illumination (dissected view, upper half of the cornea removed). *I*, Incident light passing through the cornea forms a parallelepiped and illuminates a sector of the iris surface. The opacity on the posterior corneal surface is viewed by the observer (*O*) in the direction of the retroreflected rays.

flecting screen. Similarly, a lens opacity which may appear bluish green in focal illumination, appears yellowish or dark in retro-illumination. The cornea and lens normally are relucet under direct focal illumination (with the resultant formation of optic sections

or blocks) but they are nonrelucant and nonresponsive (nonscattering of light) in retro-illumination.

No special details of the transparent media are revealed by retro-



FIG. 72. Appearance of the posterior (pigment) corneal opacity with technique shown in Figure 71.

illumination unless there is some abnormality (pathologic tissue) which obstructs, resperses, or refracts this light. Special characteristics of such structures can be observed by retro-illumination, which otherwise might pass unnoticed. With this method, in contrast to direct focal illumination, the focal point of the microscope does *not* correspond to the focal point of the beam. When the cornea is clearly in focus with the microscope, the exact focal point of the beam is on a plane behind the cornea (usually on the iris).^{*} In examining the lens by retro-illumination, it is possible to use light reflected forward from the posterior capsule. For transillumination of the iris, the beam may be directed into the lens through the pupil and the adjacent iris is observed by the light reflected from the lens. In this

^{*} This point is important when using indirect retro-illumination (page 83) when the size of the illuminated patch on the reflecting screen must be small in order to allow for an adjacent area of dark background.

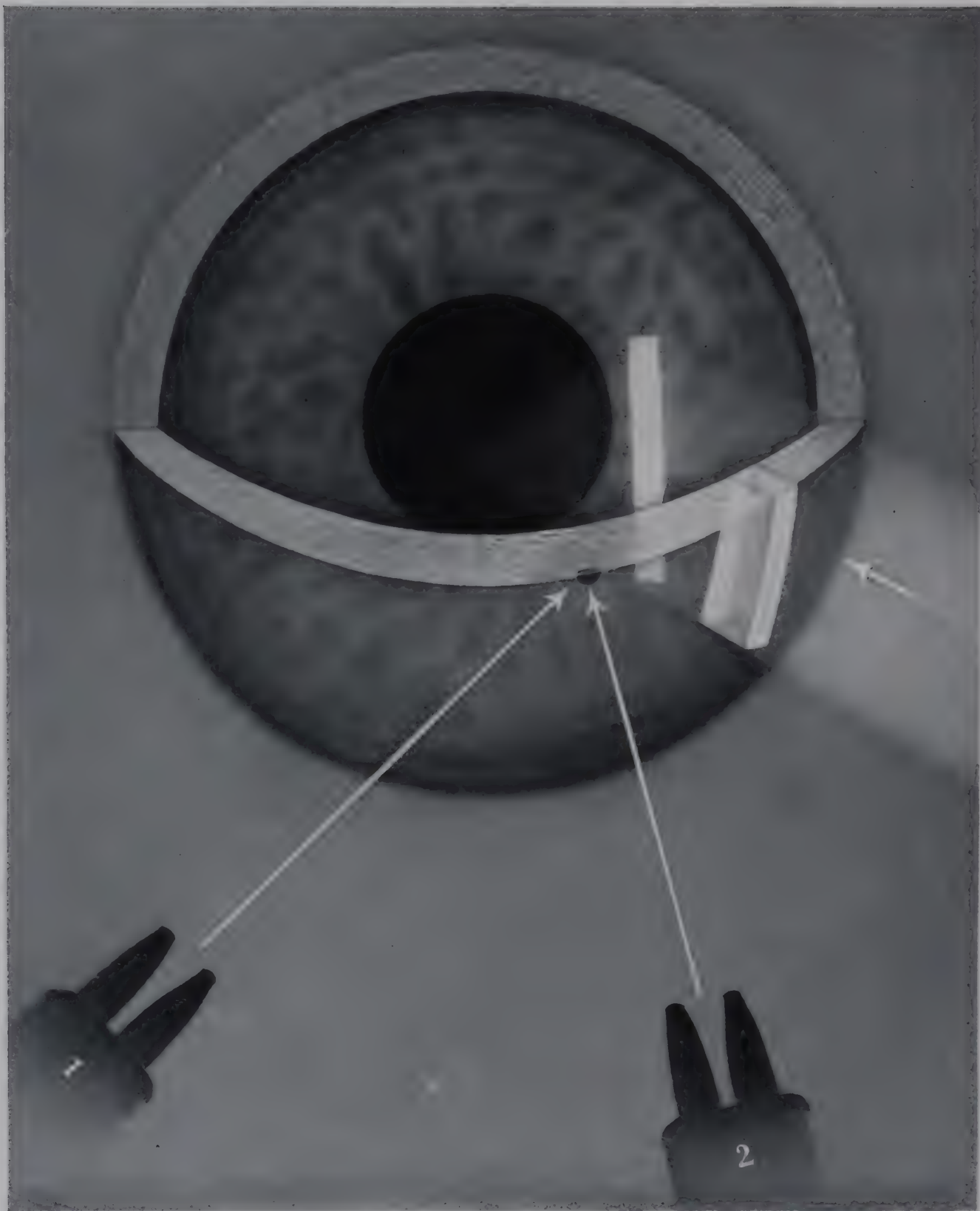


FIG. 73. Indirect retro-illumination. The position of the observer changes from position 1 to position 2 so that the opacity on the surface of the cornea is now viewed in the retroreflected light against the black background of the pupil.

case, the beam is sharply focused on the anterior lens capsule. Little, if any, of the light is able to pass through the dense tissue of the normal iris. Only an atrophic or albinotic iris can be satisfactorily retro-illuminated.

Retro-illumination is the method of choice for examination of epithelial edema, vacuoles, posterior precipitates, delicate scars, and blood vessels of the cornea.

In the lens, capsular and subcapsular changes (vacuoles) and many types of lenticular opacities can be observed. In the atrophic iris, thinning and pigmentary defects can be demonstrated. Retro-illumination is also of some value in examining conjunctival and episcleral structures.

Graves analyzed retro-illumination, especially in relation to the cornea. He classified it according to the manner in which it is affected (1) by the angles between observation and the reflected beam,* and (2) by the dominant optical properties exerted by pathologic tissue as revealed by retro-illumination.

(1) *Direct retro-illumination* is obtained when the observed feature is viewed in the direct pathway of the reflected light. In this case the area of the illuminated background is directly behind the observed structure (Figs. 74, 75, 76).

Indirect retro-illumination results when the observed feature is not viewed in the direct pathway of the reflected light. Here the retro-illumination object is viewed against a dark nonilluminated background. In other words, the reflecting illuminated surface is to one side of the axis of observation (Figs. 77, 78, 79, 80).

* This sometimes is known as positive and negative darkfield illumination and has been described in the catalogue of the Zeiss Company as follows: "This method consists neither in directing the pencil of light upon the actual area under observation nor in viewing this area by the indirect light of the brightly illuminated and sharply defined focal area in its immediate neighborhood; the light is concentrated more or less behind the area of tissue that is to be examined, and observation is made by light reflected back from the focus. No single ray from the illuminating system can therefore enter directly into the microscope, the image being formed exclusively by diffracted and scattered light. The axis of the microscope must be approximately at right angles to the axis of illumination. If this angle is more obtuse than a right angle, what is termed negative bright field illumination is obtained, the particles appearing dark on a bright ground, whereas in positive darkfield illumination they appear bright on a dark field. Darkfield observation is less trying to the eyes than other methods, and does not give rise to entoptic phenomena which are sometimes troublesome when observing by direct light."

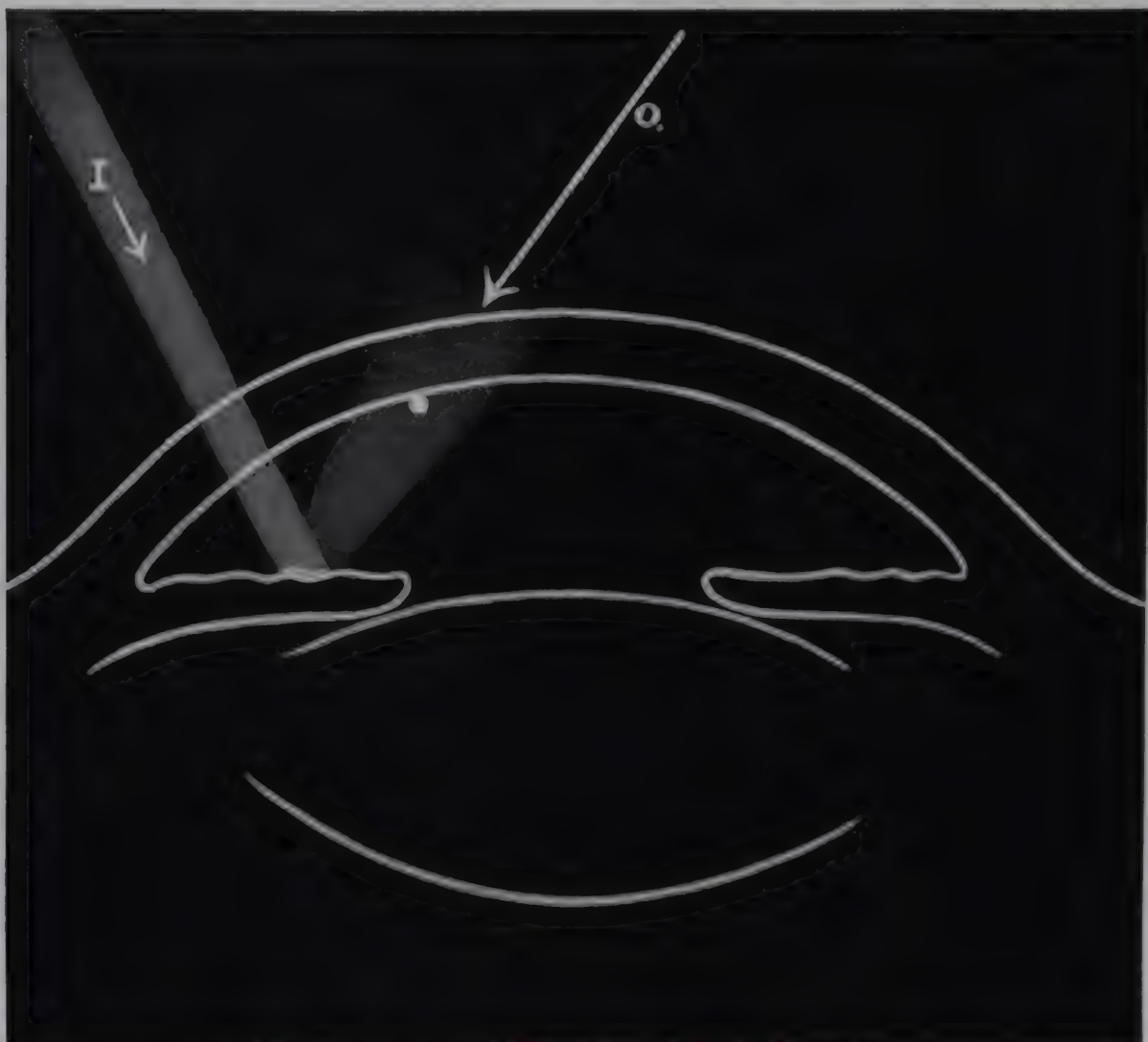


FIG. 74. Diagram illustrating direct retro-illumination of a deposit on the posterior corneal surface. O, Observer; I, incident light. The angle of observation corresponds to the angle of the light reflected from the iris surface.



FIG. 75. The appearance (partially obstructive) of a deposit on the posterior corneal surface as seen by direct retro-illumination. Note halo of light completely surrounding the deposit.

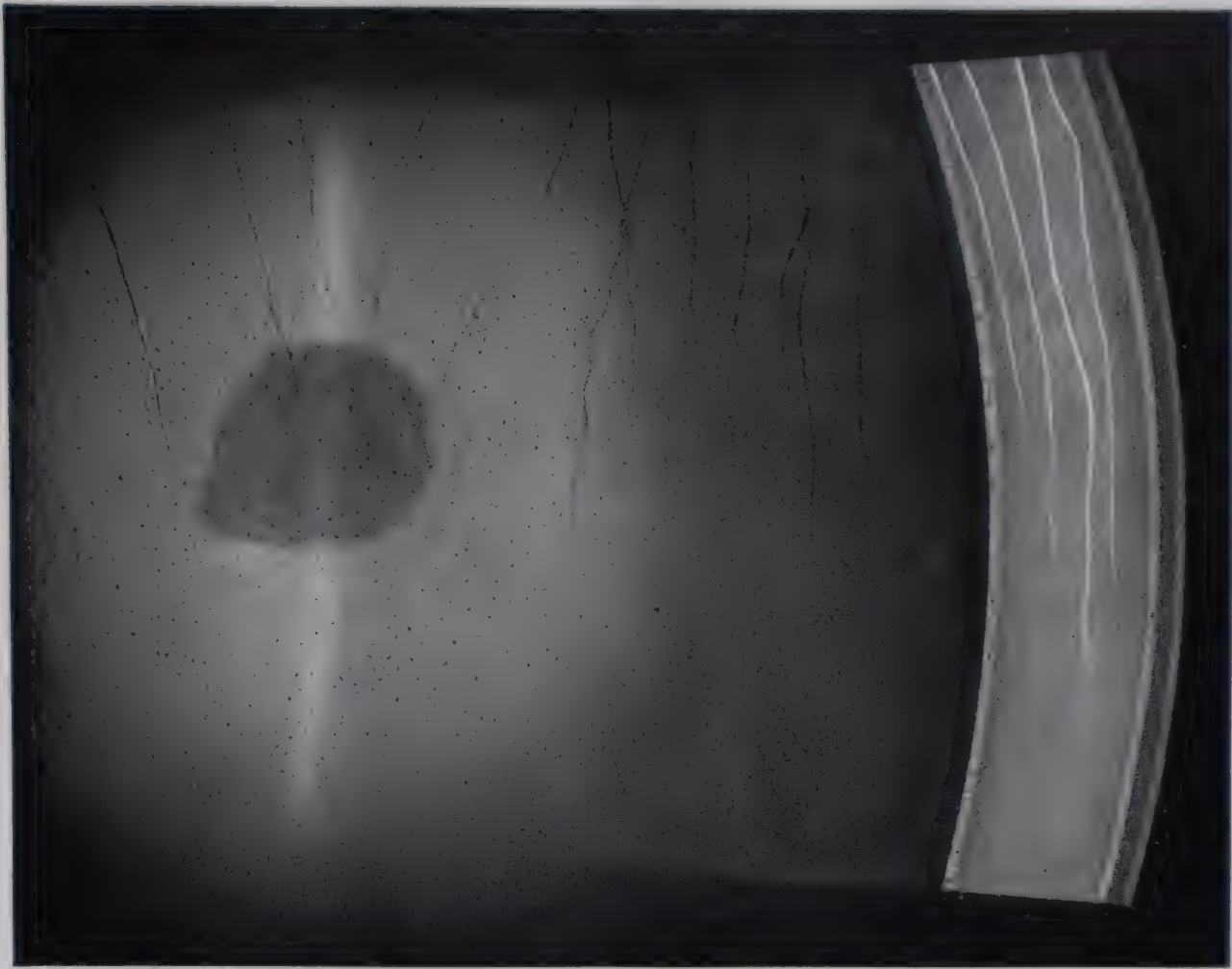


FIG. 76. Corneal vascularization in optic section and by direct retro-illumination. In the section the vessels appear as white lines, and pigment granules are localized on the posterior corneal surface. To the left the pigment granules, scar, and vessels which are obstructive appear dark against the background of the illuminated iris (direct retro-illumination).

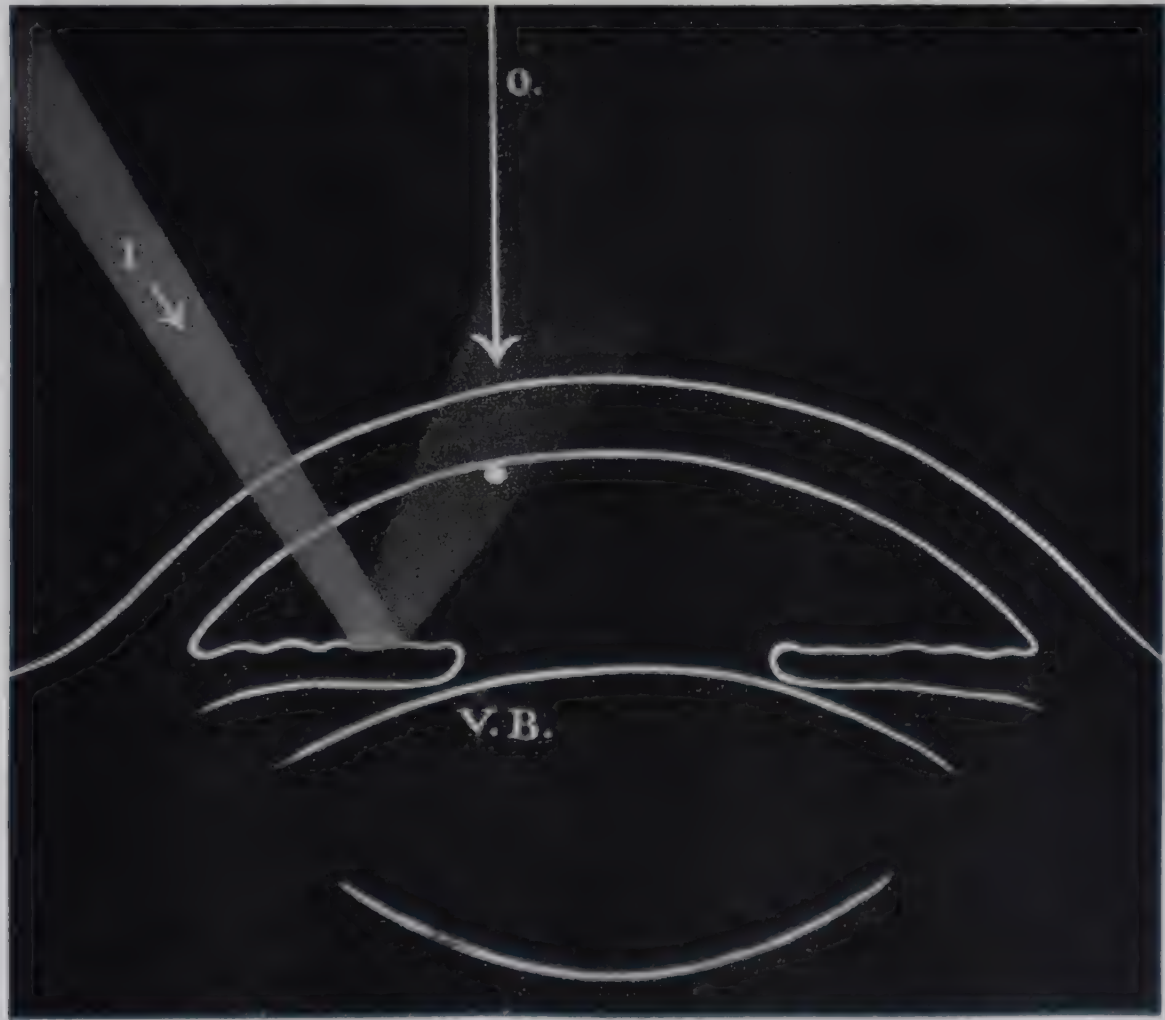


FIG. 77. Indirect retro-illumination. Diagram showing a deposit on the posterior corneal surface as viewed by indirect retro-illumination. The angle of observation (O) does not correspond to the direction of the reflected rays, but is such that the retro-illuminated object is viewed against the dark nonilluminated background (VB) of the pupil.



FIG. 78. Indirect retro-illumination. The actual appearance of a deposit as seen by indirect retro-illumination. (The deposit is somewhat respersive and illustrates reversed illumination; that is, the margin of the deposit away from the light is luminous.)



FIG. 79. Keratic precipitates by direct focal illumination, direct and indirect retro-illumination. The precipitates by indirect retro-illumination show crescentic halos of light on the side farthest away from the light (reversed). Note that the keratic precipitates which appear solid white by direct focal illumination are dark by retro-illumination.

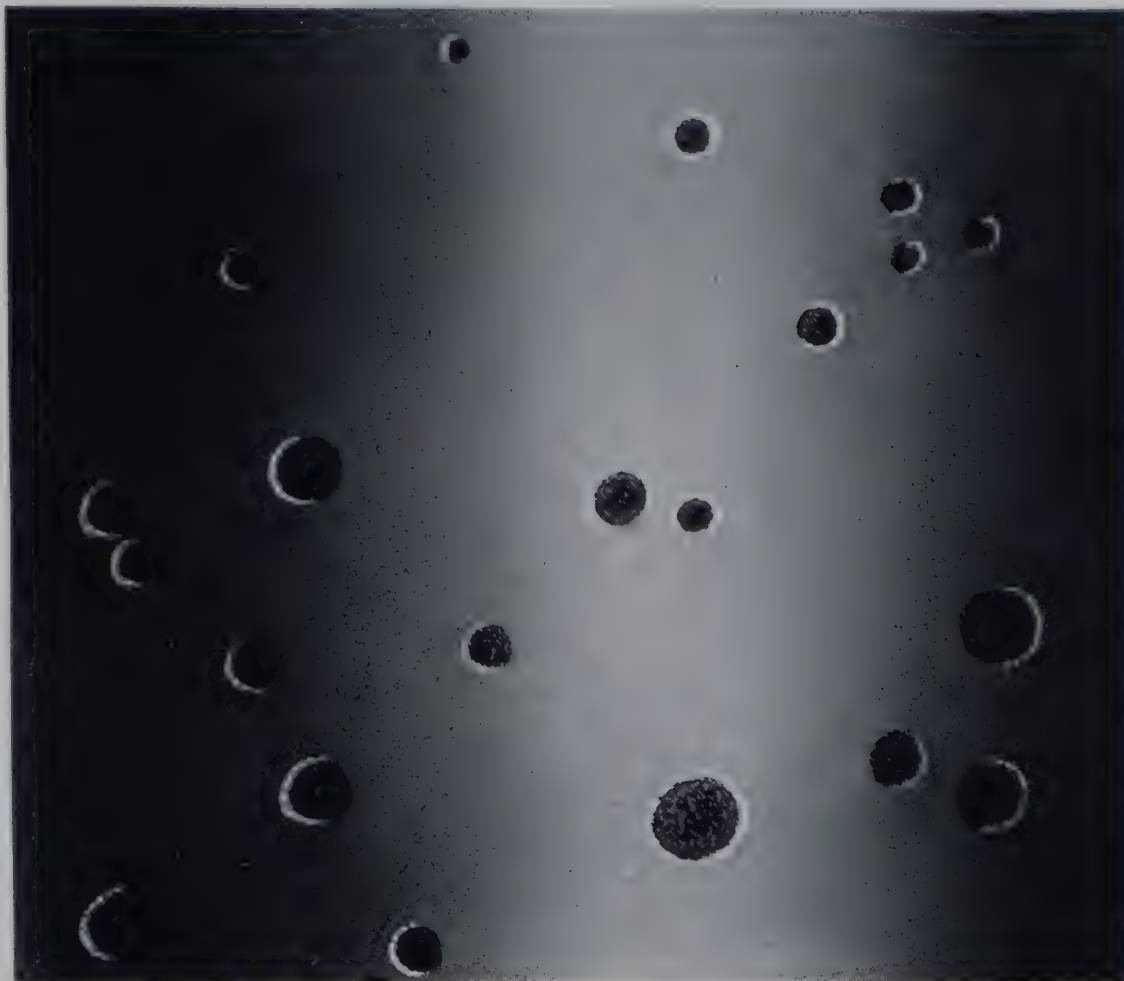


FIG. 80. High power of Figure 79, direct retro-illumination and indirect retro-illumination. The deposits by indirect retro-illumination show reversed illumination; the particles directly in front of the illuminated iris are partly obstructive as indicated by a faintly stippled appearance.

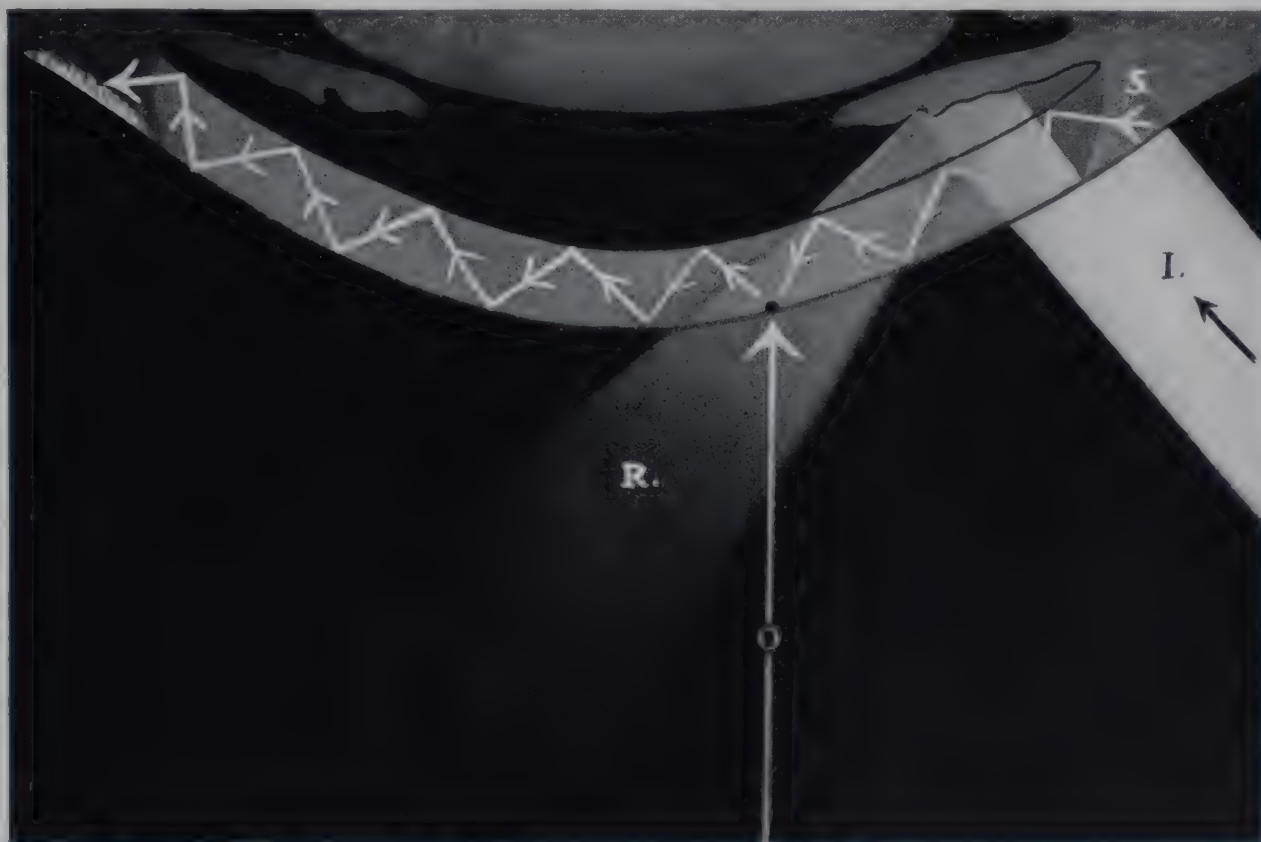


FIG. 81. Diagram illustrating the pathways of light and direction of observation in combined sclerotic scatter and indirect retro-illumination. *I*, Beam; *R*, path of retro-illuminated beam; *O*, observer; *S*, direction of scattered light.

A combination of direct and indirect retro-illumination and sclerotic scatter is obtained by placing the beam so that it falls partly on the sclera at the limbus and partly on the iris (Fig. 81). By this



FIG. 82. The refractile effect of a large vacuole of the corneal epithelium when observed in front of the junction of the illuminated iris and the dark pupillary background. The iris border appears distorted when viewed through the vacuole.

procedure an opacity may be viewed by retro-illumination and sclerotic scatter.

(2) *Optical properties of structures examined in retro-illuminated transparent media:* (a) obstructive — opaque to light; (b) *respersive* — scatters light; and (c) *refractile* — refracts, minimizes, or distorts the view of the background.

(a) The *obstructive property* is seen in pigment or vessels containing blood, which, being opaque, obstruct the light and appear dark against a bright background (Figs. 75 and 76).

(b) Pathologic changes which scatter but do not completely obstruct light when retro-illuminated are *respersive* (e.g., edema of the epithelium, corneal precipitates) and, therefore, look brighter than the dark background. Certain corneal lesions, such as, infiltrations or nebulæ which are whitely opaque (*relucent*) in direct focal illumination, may appear by retro-illumination to be composed of groups of delicate droplets (Fig. 209).

(c) Because the refractive indices of vacuoles, empty vessels, and deposits differ from refractive indices of the media in which they lie, certain refractile illumination effects are obtained when the beam is properly directed (Fig. 82). By refraction these effects distort or minify (Graves) the view of the junction of the illuminated and unilluminated areas of the background. To obtain this junction when examining the cornea, the beam is directed in such a way that the pupillary margin of the iris is illuminated without allowing the direct focal illumination of the lens to interfere with the adjacent dark contrasting background (Figs. 83, 84).

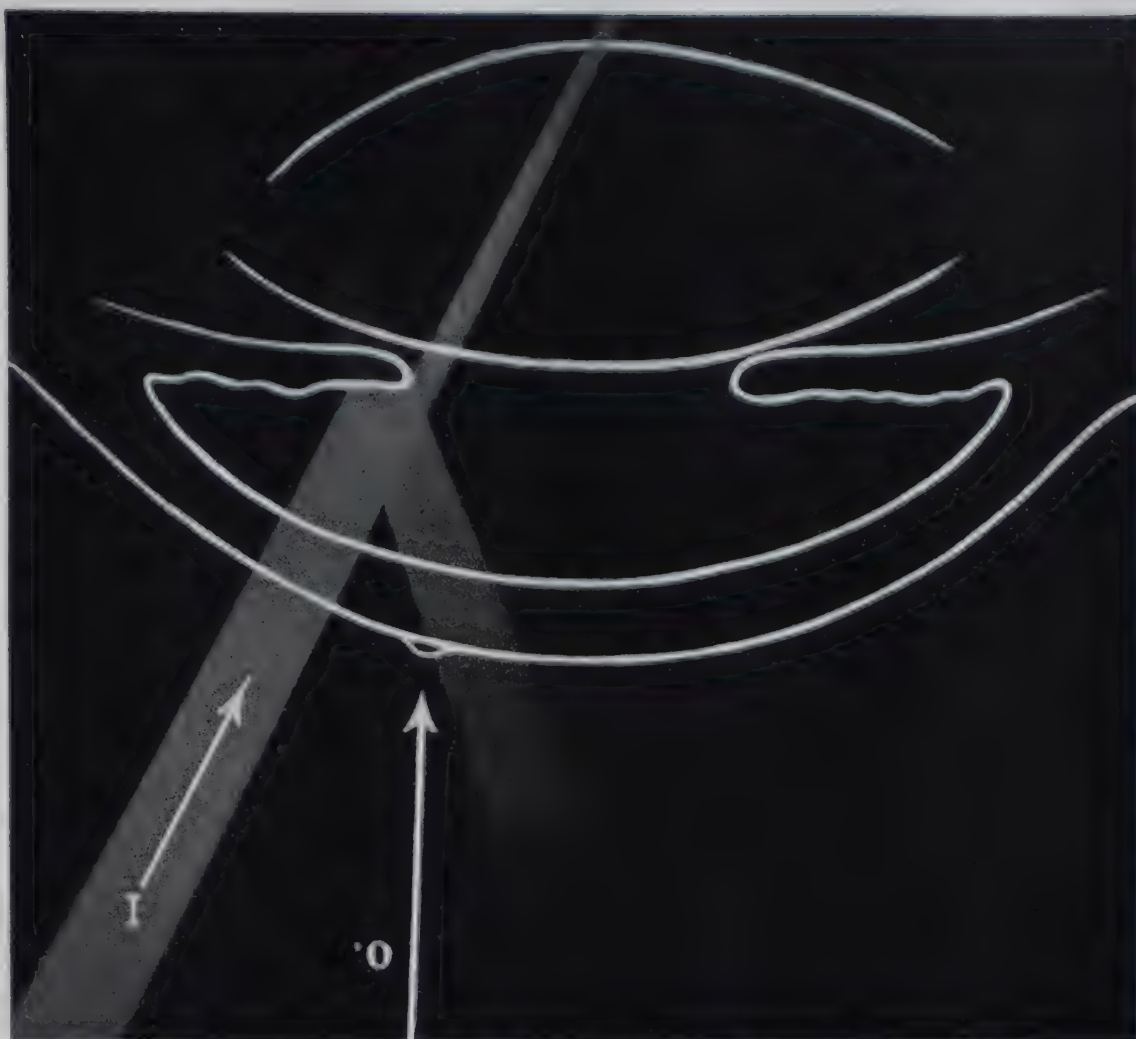


FIG. 83

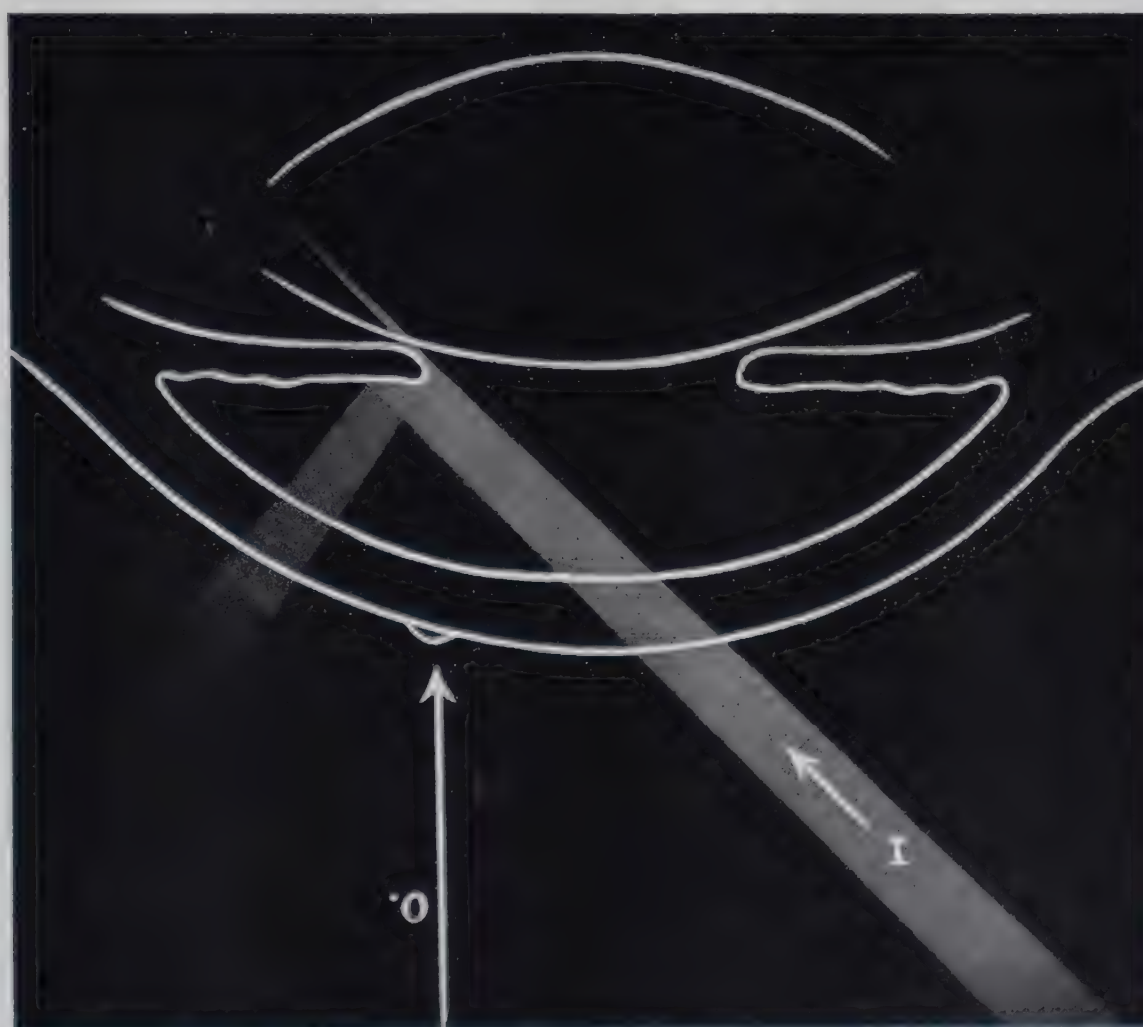


FIG. 84

FIGS. 83 and 84. Retro-illumination of the area of the cornea opposite the pupil margin. In Figure 83 the ability of the pupil margin to confer a sharp border to the illuminated area of iris is impaired to some extent by the associated illumination of the area of the lens within the visual background. This disadvantage is avoided in Figure 84.

In addition to these phenomena, vacuoles and precipitates, when viewed by retro-illumination, may exhibit other properties. In direct retro-illumination a vacuole is bordered by a dark line in the form



FIG. 85. Lens vacuoles by direct retro-illumination and indirect retro-illumination. By direct retro-illumination the vacuoles have a dark border around them. By indirect retro-illumination the vacuoles display unreversed illumination, that is, the margin of the vacuoles nearest the light is outlined by a bright crescent of light.

of a ring or a crescent (Fig. 85). By indirect retro-illumination the vacuole may display unreversed illumination; that is, the margin of the vacuole nearest the light is outlined by a bright crescent of light while the opposite side is dark. On the other hand, a solid precipitate shows the opposite of this; that is, reversed illumination. In this case, the margin away from the light becomes more luminous.

To recapitulate, direct retro-illumination is most useful in observing structures that obstruct or refract light, while indirect retro-illumination is recommended for the study of structures that are refractile or respersive. Vacuoles display "unreversed illumination" by indirect retro-illumination, that is, the side of the observed feature nearer the light is brighter than the remote side. Solid or opaque precipitates reveal "reversed illumination." Thus, information can be obtained concerning not only the form of structures but also their refractive index as compared with the refractive index of the surrounding media.

Localization in retro-illumination. As previously stated, the light of retro-illumination is somewhat diffuse and, therefore, no block or optic section is formed. Consequently, in order to localize a structure by means of this method, differences of focal depth, stereoscopic observation, or comparison with other structures the depths of which are known, must be employed. As in Plate XXIV, fig. 3, the vacuolar carpet of epithelial edema is observed in retro-illumination and, then, by racking the microscope forward (i.e., deeper) the changes on the posterior face (deposits) come into view, illuminated by the same retroreflected rays (Plate XXV, fig. 4). Light reflected from the posterior surface of the cornea and lens may be utilized as a source of light for retro-illumination of structures situated more anteriorly. In most instances, after details are observed in retro-illumination, they are then viewed and localized in direct focal illumination (optic section).

ZONES OF SPECULAR REFLECTION

When passing the focused beam across the surfaces of the cornea and lens one is struck by the brilliancy of the light reflexes. The reflex from the anterior corneal surface is especially dazzling. The smooth surfaces of the cornea and lens act like mirrors.

In the early days of ophthalmoscopy these reflexes, especially those from the anterior corneal surface, were annoying because they interfered with the observation of the fundus. However, the modern ophthalmoscope is so constructed that specular reflexes are largely avoided. Proper use of these mirror reflexes in biomicroscopy permits observation of surface details which cannot be seen by other means.*

Zones of Discontinuity. In addition to other phenomena (transmission, absorption, fluorescence, polarization), reflection of light occurs when a beam of light is incident on an optical surface whose

* It is interesting to note that Tscherning²⁹⁸ spoke of being able to study details of the anterior lens capsule in "reflex." However, it was not until Vogt stressed the importance of discriminating between the zone of specular reflection (the plane where regular reflection occurs) and the image of the luminous source that the clinical significance of this subject was appreciated. At each major zone of discontinuity in the eye, it is possible to differentiate a zone of specular reflection and an image of the luminous source.

refractive index differs from that through which the light has passed. Such a surface is known optically as a zone of discontinuity.

In the eye there are many such zones of discontinuity, which act as mirror surfaces. Chief of these are the anterior and posterior surfaces of the cornea and the lens; less marked zones may be demonstrated within the substance of the cornea and lens. In the chief zones, the greater differences of the refractive indices and the higher polish of these surfaces enhance their mirror-like properties.

Regular Reflection. According to the laws of optics, when a beam of light is incident on a mirror surface, part of the beam is subject to regular reflection in which the angle of reflection equals the angle of incidence, both lying in the same plane.

Owing to surface irregularities another part of the beam is reflected in a diffuse irregular manner. The regularly reflected portion permits one to see an image of the illuminant, while the irregularly reflected portion permits one to discern details of the reflecting surface itself. It is well known that the reflecting properties of a silver mirror are partly dependent on the smoothness of the surface. The smoother and more highly polished the surface the more difficult it is to discern the actual zone at which reflection occurs. This zone can be seen only if there are present surface irregularities, which cause irregular scattering or reflection and give rise to secondary images, that are visible within the zone * (Fig. 86).

If it were possible to construct an ideally perfect mirror all light incident on its surface would be completely reflected in a regular manner and it would be impossible to see its surface. However, in nature no such ideal surface exists since even the most perfect mirrors have microscopic irregularities, and consequently, varying degrees of irregular reflection from surface irregularities occur, which permit observation of the surface or the place where reflection occurs.

The following is an illustration of regular reflection: If a beam of sunlight (parallel rays) enters a darkened room through a crack

* As Koby¹⁷⁵ pointed out, "these irregularities produce in their turn secondary, catoptric images, the collection of which constitutes the zone of specular reflection which our microscope will perceive, the radius of curvature of these secondary inequalities being so small, and the images, therefore, being very near to the primary surface."

and falls on a piece of polished glass on the floor, the rays are reflected (the angle of incidence equalling the angle of reflection) against the wall where a patch of light is seen (image). The glass

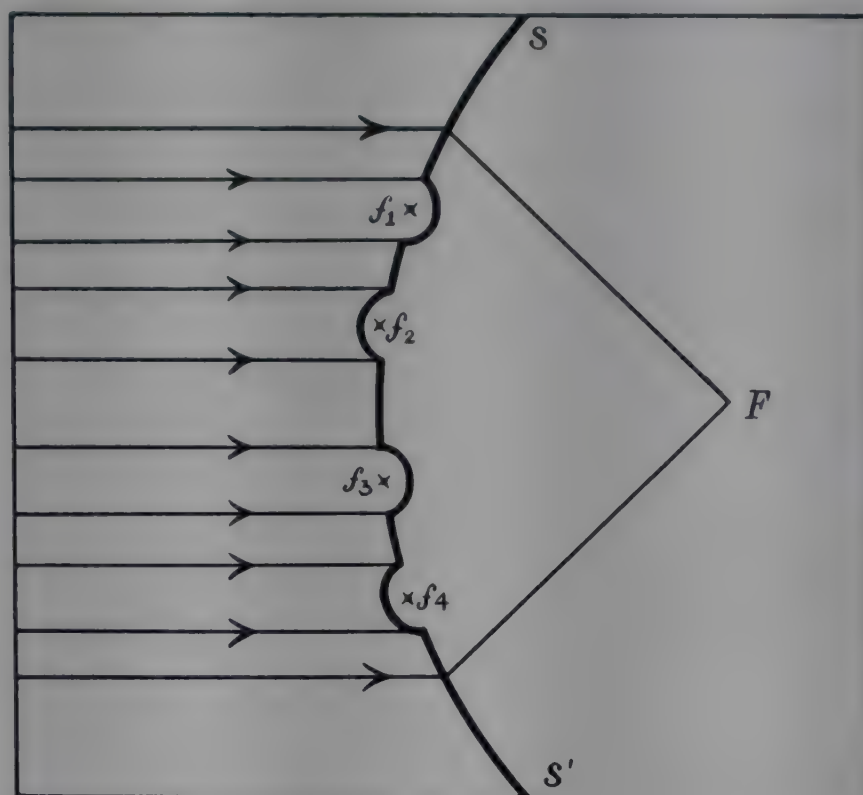


FIG. 86. Diagrams of the zones of specular reflection. A primary surface, SS' , struck by a beam of parallel rays will form a primary image (here virtual) of the luminous source at F . Little irregularities of surface, alternately convex and concave, will give secondary images at f_1, f_2 , etc., situated very near to the primary surface. These images together constitute the zone of specular reflection. (After Koby.)

(reflecting surface) is not visible except when the eye of the observer is in the direct path of the reflected beam. When this occurs the glass suddenly stands out as a dazzling object. This dazzling reflex is produced by regular (specular) reflection. The amount and intensity of light reflected are directly proportional to the smoothness of the reflecting surface and the angle of incidence of the light beam. Irregularities, such as dust, on the surface of the glass, are discernible because they reflect light irregularly from the surface at which regular reflection occurs. Defects produced by imperfect polish, reflect little or no light but rather absorb light and, therefore, appear as dark spots in the plane of the dazzling surface (Fig. 87).

Irregular Reflection. Irregular diffuse reflection occurs throughout nature and accounts for the visibility of most nonluminous surfaces and objects. The dancing shimmer on the apparently smooth

surface of a quiet pool in bright sunlight is caused by the reflection of the sun's rays into the eyes of the spectator from countless little ripples on the surface of the water.

Zones of Specular Reflection as Seen in the Normal Eye. Both types of reflection (regular and irregular) are observed at each zone of discontinuity in the eye. Regular reflection at the mirror-like zones is called "specular reflection" and can be seen only when the observing eye is placed in the path of the regularly reflected beam (angle of reflection equalling angle of incidence), thus receiving the specular reflex (Figs. 87, 88). The color of the light source determines the color of the specular reflex.

On the surface of the cornea this phenomenon of specular reflection is easily seen as a dazzling reflex. The actual surface area at which the reflection takes place is called "the zone of specular reflection." The surface irregularities in this zone give rise to irregular reflections which produce secondary images and cause the surface plane to become visible. When viewed with the microscope, the zone of specular reflection for any given position occupies only a small portion of the surface of the parallelepiped because the corneal curvature limits the visible area in which it is possible to perceive the specularly reflected rays along one observation axis.

Surface elevations or depressions appear as dark defects in the brilliant zone of regularly reflected light owing to the fact that the light reflected from them is irregular or diffuse, thus forming secondary images situated very near to or at the surface plane. However, if some of the rays (e.g., from the summit of an elevation or from the depths of a depression) are reflected back along the axis conforming to specular reflection, they are seen as shining glints in the center of the dark areas (Figs. 89, 90).

Certain points must be considered in carrying out the principles of observation by specular reflection in the cornea. To understand the optical phenomena occurring at each zone of discontinuity one must discriminate first the effects of diffuse reflection which forms the corneal parallelepiped; second, the catoptric reflex of the illuminating lens which is seen in the anterior chamber to one side of the parallelepiped (visible from any angle); and third, the

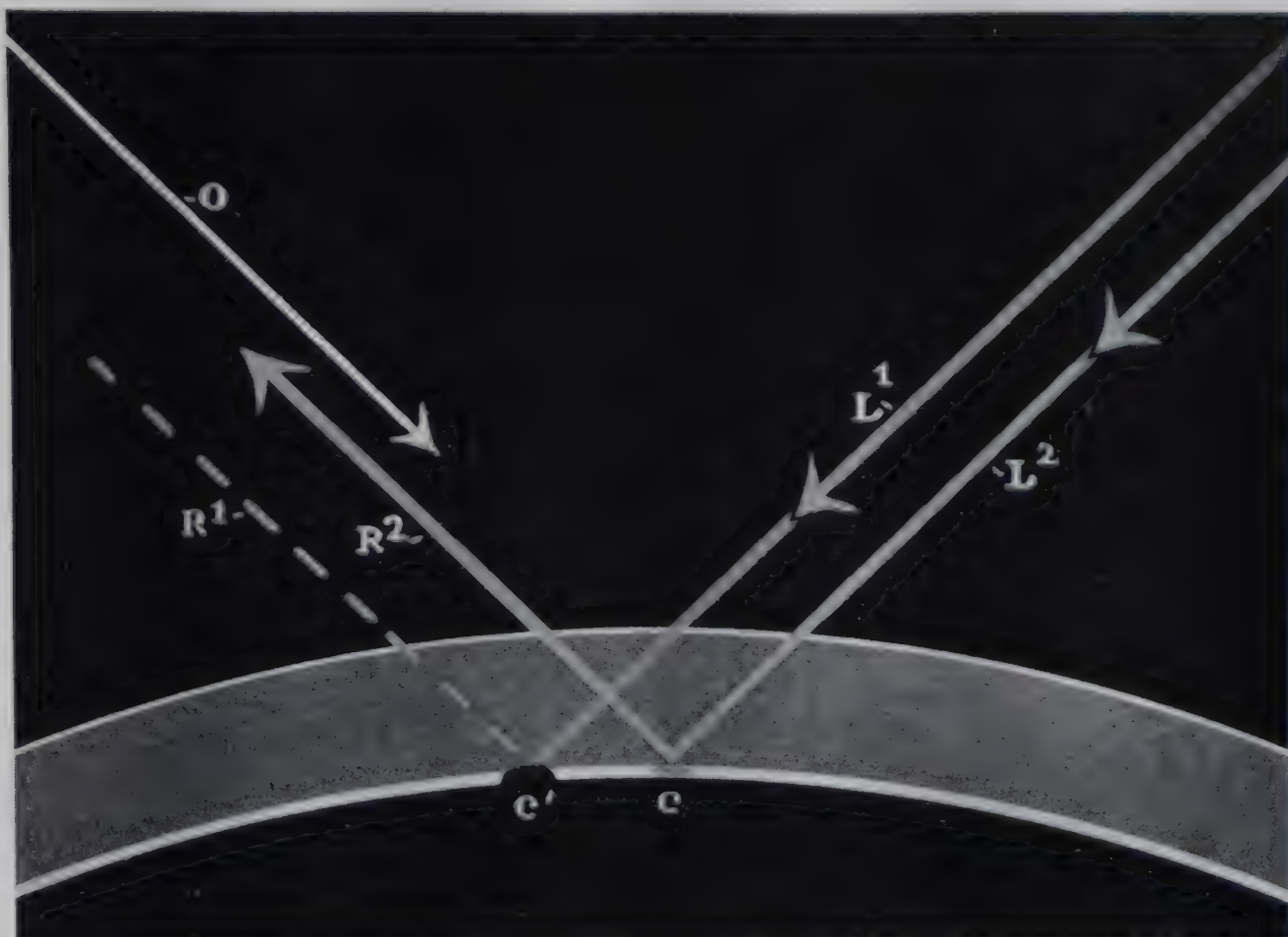


FIG. 87. Regular reflection occurs at a point C on the posterior corneal surface and is observed along the direction (O) when the angle of observation is equal to the angle of incidence. Beam L^1 impinges on area where there is a defect (C'). Consequently, no light will be reflected at this point (C') and it will appear dark in the surrounding specular areas. This would correspond to the imperfections in polish of a mirror.

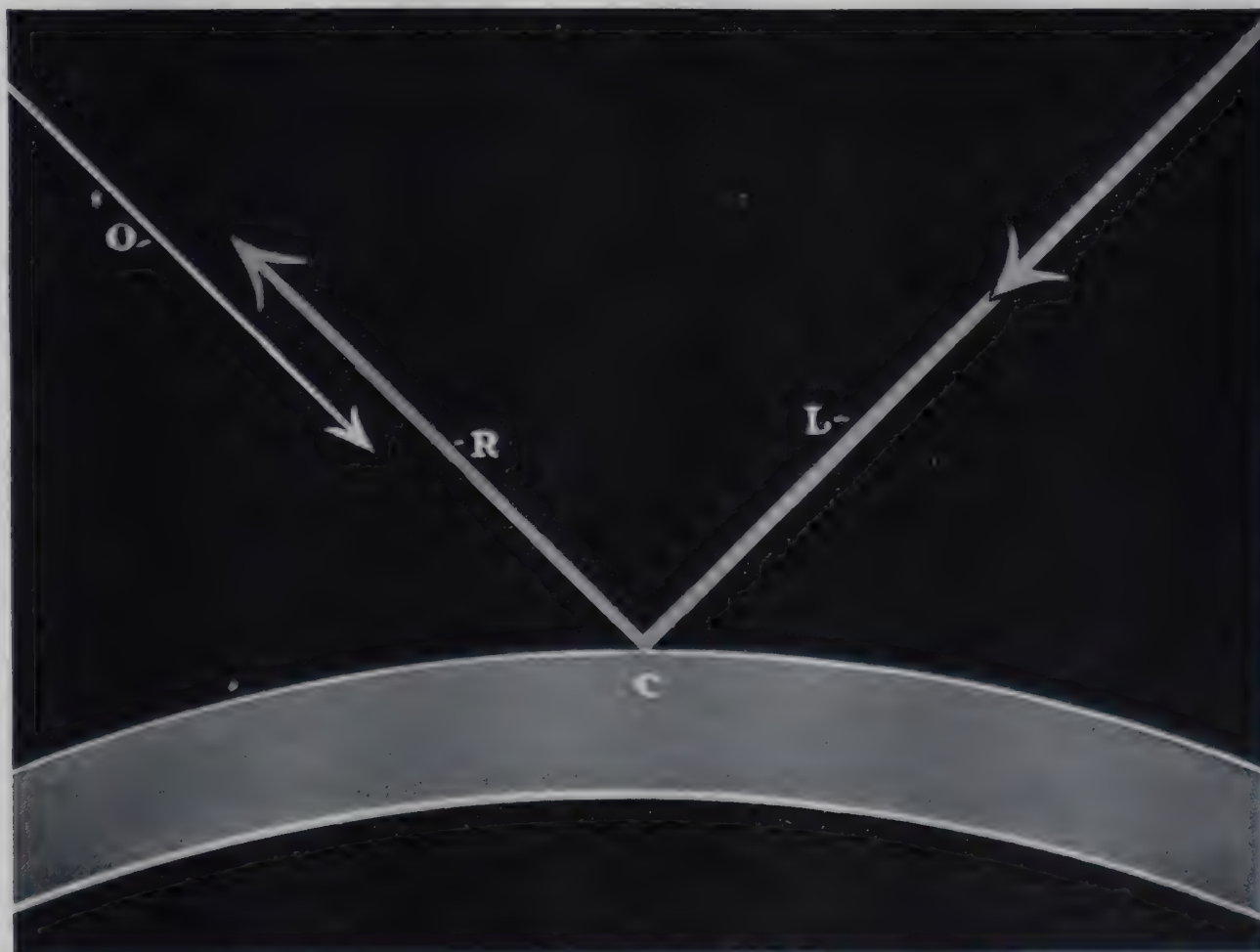


FIG. 88. Regular reflection on the corneal surface at point C can only be seen if the observer's eye (O) is in the direction of the regularly reflected rays. This means that the tangent perpendicular to the point C will produce equal angles (angle of incidence equals the angle of reflection).

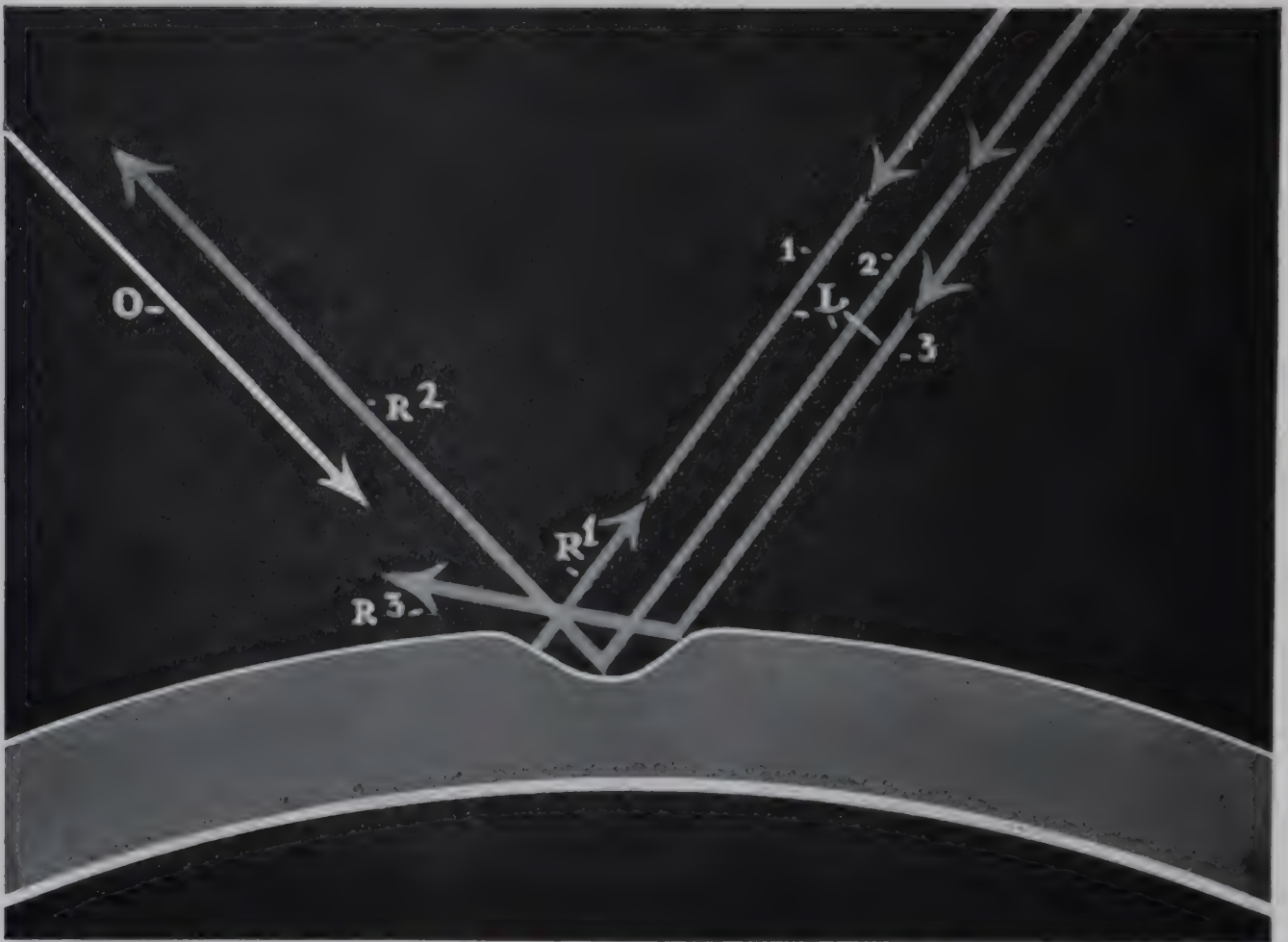


FIG. 89. The effect of a depression in the specular surface. The depression will appear as a dark defect in the brilliant zone of regularly reflected light because such rays, L^1 and L^3 will be reflected in an irregular manner as R^1 and R^3 and consequently will not be seen along the observation axis (O). However, if a ray, such as L^2 , striking the bottom of the depression is reflected back along the axis conforming to specular reflection, it will appear as a shining glint in the center of the dark area.

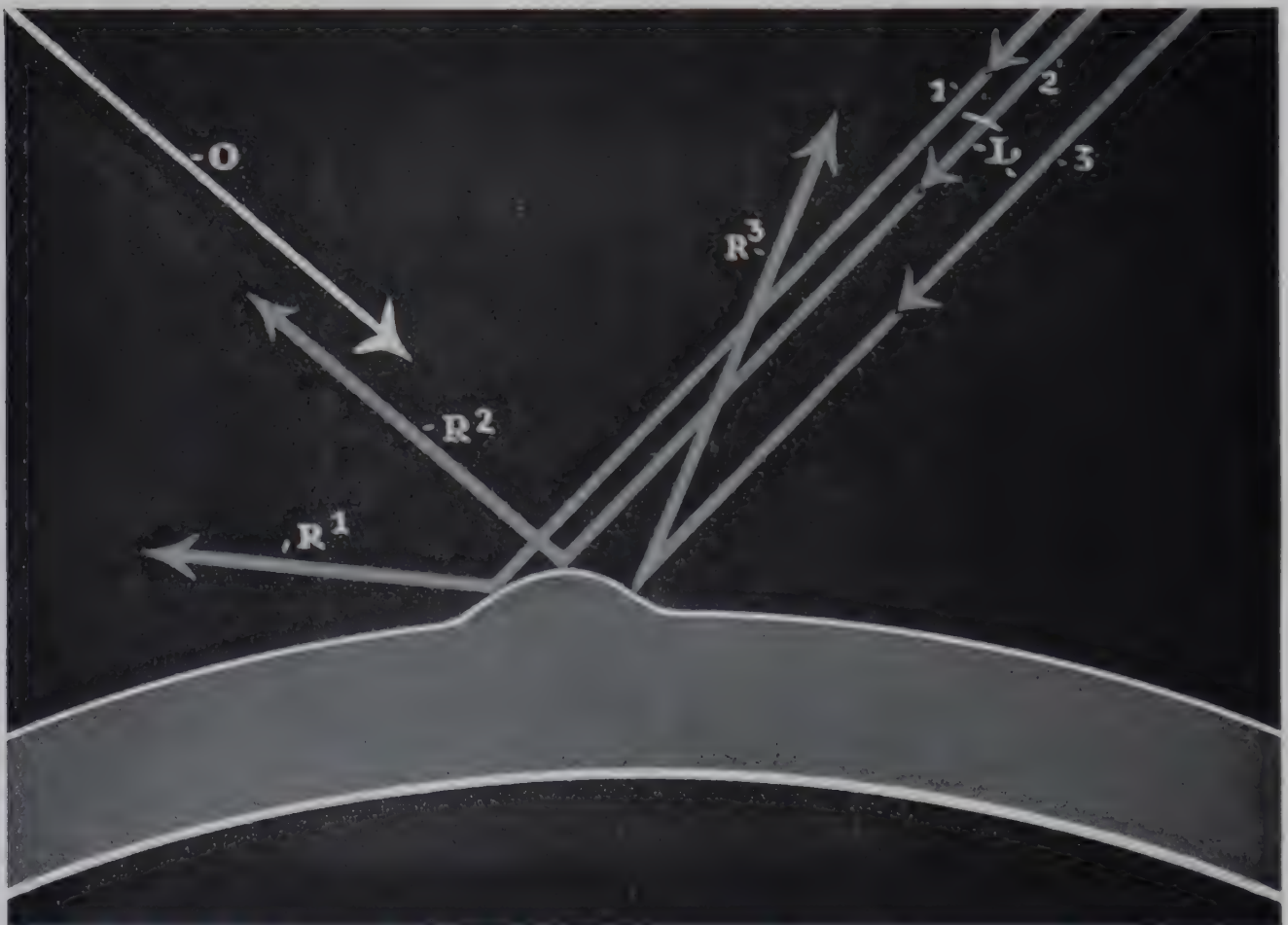


FIG. 90. In the case of an elevation, rays L^1 and L^3 will be reflected irregularly in the direction of R^1 and R^3 and appear dark along the observation axis (O). Whereas L^2 striking the summit of the elevation will be reflected in the direction of R^2 and will appear as a glint in the center of the dark area because this reflected ray conforms to the axis of specular reflection at this point.

brilliant dazzle of specular reflection (caused by regular reflection from the anterior corneal surface). When the microscope is focused exactly along the axis of these dazzling rays on the anterior surface of the parallelepiped, the zone of specular reflection is brought into view by the light reflected irregularly from tiny surface irregularities. Furthermore, it will be noticed that as the focus of the microscope is carried slightly deeper to the zone of specular reflection and along the same axis, an image of the luminous source will be seen as a coiled filament diaphragmed by the illuminating lens in the Kohler-Vogt system. Similarly, the image of the filament may be observed in the depths of the lens when the slit beam is focused on the anterior lens capsule. In the Koeppe-Poser models the presence of a ground glass in the condensing system obliterates any image of the coiled filament.

The zones of specular reflection of the corneal surfaces are obtained by the following procedure: (1) The patient's gaze is directed about 30 degrees to his right. (2) Without employing the microscope the beam is directed on the left cornea from the temporal side and the parallelepiped* is brought into sharp focus at a point about 3 or 4 mm. from the nasal limbus. (3) To the observer's left of the parallelepiped either the lens block is seen (i.e., if the light enters the pupil) or an illuminated portion of the iris. (4) To the observer's right of the parallelepiped and apparently in the anterior chamber, a whitish rectangular reflex is seen, containing the catoptric image of the illuminating lens (Fig. 91). When the parallelepiped is in sharp focus, this catoptric image is not sharp; but if the image is brought into sharp focus, it becomes smaller and its outline sharply rectilinear. If the illuminating lens is unclean, the reflex may have a grained appearance. Focusing still deeper, the image of the coiled filament may be seen if the Kohler-Vogt system of illumination is employed. In the Koeppe and Koeppe-Poser systems of illumination the image of the coiled filament cannot be seen because of the ground-glass diffusing plate in the condensing system (Figs. 92, 93). (5) Even with the un-

* In order to obtain a larger and more intense reflecting surface it is advisable to use the full width of the beam and to direct it with a wide angle of incidence.



FIG. 91. Relationship of corneal parallelepiped to the catoptric image when obtaining specular reflection from the corneal surfaces. A. Beam focused toward the nasal side of the cornea, left eye; catoptric image of the illuminating lens is to the observer's right of the corneal parallelepiped. To the left is the illuminated section of the iris. B. Moving the beam temporarily (to the observer's right) causes the corneal parallelepiped to approach the catoptric image. C. If the parallelepiped moves in front of the catoptric image the dazzling reflex of the corneal surface and the zones of specular reflection come into view. The small catoptric image is still seen to the left deeply within the anterior chamber behind the parallelepiped.

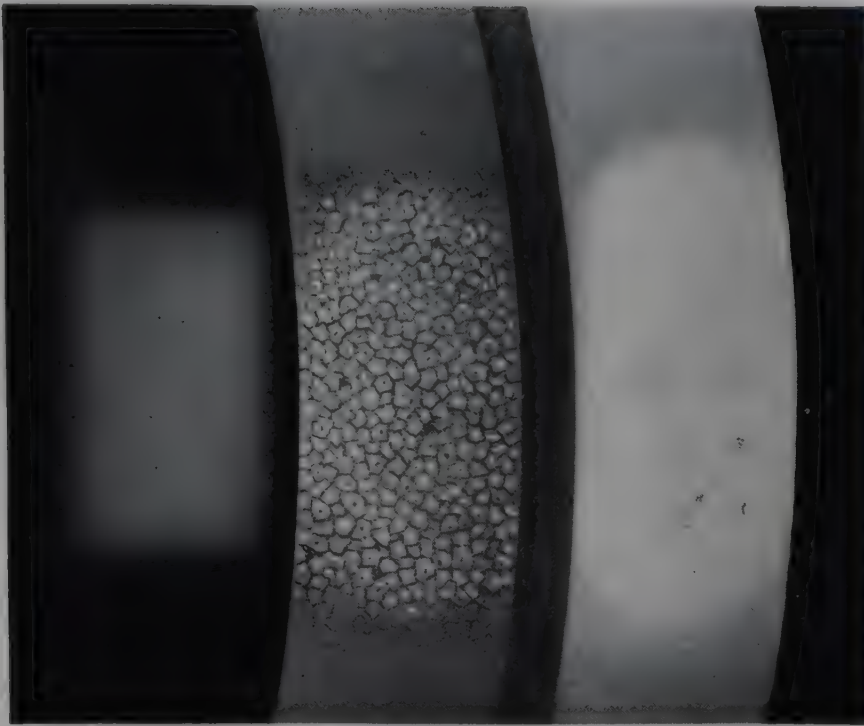


FIG. 92. High power view. Details seen in zones of specular reflection of the anterior and posterior corneal surface. The catoptric image to the left from an illuminating lens is situated deeper in the anterior chamber and is out of focus.



FIG. 93. Deeper focus on the catoptric image reveals an image of the coiled Tungsten filament light source. The parallelepiped and the details of the zones of specular reflection are now out of focus. The Koeppe-Poser systems, owing to the intervention of a ground-glass diffusing disk, do not reveal the coiled tungsten filament.

aided eye, if the beam is now moved to the observer's right so that the parallelepiped approaches this image, the observer is suddenly dazzled. This occurs because at this moment, the angle of incidence of the beam equals the angle of reflection and the observer's eye is exactly in the path of the reflected beam. (6) Repeating *step 5* while using the microscope (low power), as the dazzling reflex comes into view on the surface of the parallelepiped, it lies in front of the larger whitish rectangular reflex which appears irregular and out of focus and seems to lie deeper in the anterior chamber. This smaller dazzling reflex on the anterior surface of the parallelepiped is seen to occupy only a small part of the parallelepiped surface. This reflex is caused by regular reflection coming from the corneal surface. When the microscope is focused exactly on the anterior surface of the parallelepiped, that is, on the plane and place where the reflex is formed, the zone of specular reflection is observed. At this site details of the precorneal film, mucus, meibomian secretion, and the corpuscular elements of the tears are seen by the light which they irregularly reflect or absorb (Fig. 94). No details of the underlying epithelial cells are observed, because it is the thin film of tear fluid which in itself acts like a mirror. It must be emphasized that this method of illumination discloses details of the mirror surface only. (7) At the same time, and with the medium or high power, when the observer looks deeper in the parallelepiped to the left, at the level of the posterior surface of the cornea, a smaller, less luminous golden glow (specular reflex of the posterior corneal surface) is seen, which, when sharply focused, discloses the mosaic-like pattern of the corneal endothelium. At times, it is necessary to move the beam slightly from side to side in order to bring both areas into view. As has been pointed out, the reflection from the posterior corneal face is less luminous than that from the anterior surface because the difference of indices of refraction between aqueous and cornea is less than that between air and cornea. In addition, some of the light reflected forward from the posterior surface is absorbed or reflected as it passes through the corneal substance. Koby believes that the yellowish tint of the posterior corneal reflex is due to the fact



FIG. 94. Details of anterior and posterior surface of the parallelepiped in specular reflection. The brilliant zone of specular reflection on the anterior corneal surface shows corpuscular elements of the tear fluid. The zone of specular reflection on the posterior corneal surface reveals the characteristic endothelial mosaic. The dark depressed spots in the mosaic are due to the Hassall-Henle bodies.

that the cornea absorbs chiefly rays of short wave length and that this effect is increased doubly as the rays traverse the cornea to reach the posterior surface and then are reflected back to the observer.

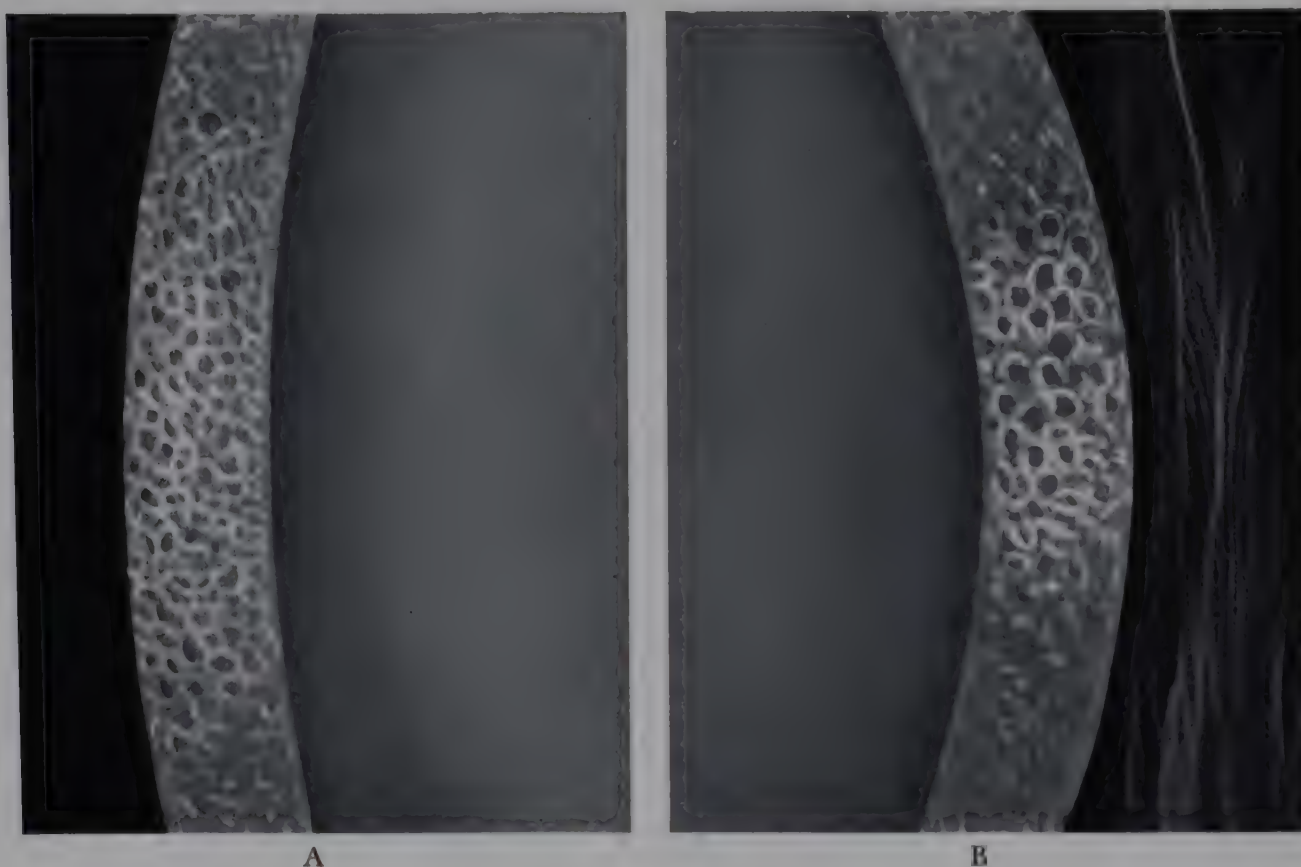


FIG. 95. The design (shagreens) on the anterior (A) and the posterior capsule (B) of the lens as seen in specular reflection.

The fact that the posterior reflex (zone of specular reflection) is smaller than the anterior is explained by the greater curvature of the posterior face which allows less rays to be specularly reflected along one given axis of observation.

Specular reflection from the anterior and posterior lens capsule can be more easily obtained. Here, the beaten-silver effect (shagreen) is observed, caused by irregularities on the surfaces of the lens capsule (Fig. 95).

The specular reflexes from the anterior surface of the cornea and posterior lens capsule are seen more easily than those from the posterior corneal surface and anterior lens capsule because of their greater luminosity. In the cornea only details of sufficient size to interfere with the normal contour of the precorneal film are visible. The posterior corneal surface reveals details more interesting than those of the anterior. The endothelium is extremely sensitive to pathologic changes occurring in the cornea. Superficial

changes in the cornea often cause endothelial disturbances. Warts, folds, and tears in Descemet's membrane and deposits on the endothelial surface, produce changes in the endothelial mosaic which are seen in specular reflection. In direct focal illumination the normal laminated structure of the corneal stroma produces an internal specular reflection (intrinsic specularity [Graves]) (Fig. 96). As the angle for viewing specular reflection from the corneal surfaces is approached, a glistening bronzed vertically striaform design is seen to form gradually in the otherwise uniformly opalescent corneal section. This scintillating design becomes more pronounced and occupies a larger area in the optic section, as the zones of specular reflection of the corneal faces are approached and entered. If one continued to move the beam in the same direction, that is, away from the surface zones (temporally in the foregoing example), the internal specularity would gradually decrease until the normal opalescence returns. Graves believes that this intrinsic specularity is due to a summation of multiple specular reflections which emanate from the laminated structural formation, of a type characteristic of the corneal structure. A similar phenomenon occurs in the lens.

Reflections from the interior of the lens result in the appearance of the successive reflecting surfaces or zones of discontinuity of the lenticular nuclei (Fig. 70). These surfaces represent the internal lenticular architecture arising from its natural stratification. In contrast with the capsule, they do not reveal well-defined zones of specular reflection because of insufficient demarcation. However, an increase in specular reflection occurs from the surface of these zones of discontinuity as senescence is approached (owing to increasing differences of refractive indices). In comparison with the adult lens the infantile lens shows little internal reflection. These zones of discontinuity, which represent the surfaces of the fetal and adult nuclei and of the capsule, were first discovered by Gullstrand.

Either by altering the angles of observation or illumination or by directing the patient to move his eye, any part of a reflecting surface can be examined. Because of the smallness of the incident beam, the corneal curvature, and the double objectives used, the reflected rays can be observed only in one ocular at a time at any given point. Al-

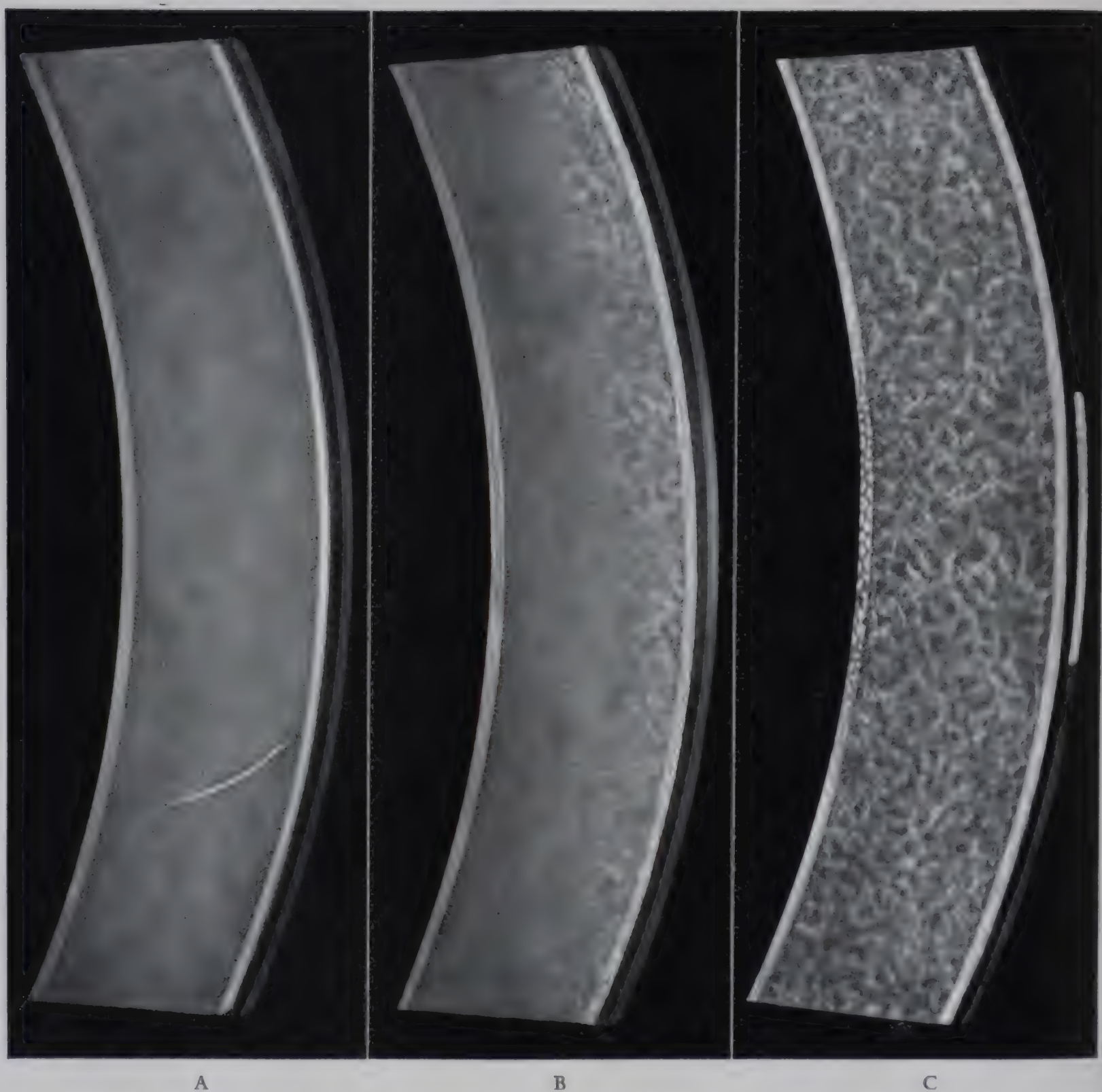


FIG. 96. Stages in the development of internal specularity of the cornea as seen in optic section. A (corresponding to position of beam in Fig. 91 A). The section displays the normal uniform corneal opalescence. B (corresponding to Fig. 91 B). The onset of internal specularity is shown in the anterior parts of the section. C (corresponding to Fig. 91 C). When the zones of specular reflection of the anterior and posterior corneal surfaces come into view, the section displays marked intrinsic specularity throughout.

ternate opening and closing of the observer's eyes will demonstrate this point. It also accounts for the fact that when observing the zone of specular reflection, if the beam is moved ever so slightly, the specular reflex is lost momentarily, only to reappear as it enters the other objective.

Reflection from Structureless Membranes. It is only when alterations occur, such as folding, that reflexes from the so-called "glass" hyaline membranes of the cornea (e.g., Bowman's, Descemet's) and from the lens capsule may be seen. These reflexes were described by Vogt, who stated that they were always linear and parallel (i.e., two for each individual fold), and that their ends become narrow and fusiform; parallax displacement occurs when the angle of observation or the angle of incidence of the beam is altered. Vogt studied the optics of these reflexes on a surface composed of parallel, alternately convex and concave mirrors. He found that the position of the reflex lines depends on the relation between the angles of incidence and observation (Figs. 97, 98). The displacement of the lines depends on the curvatures of the mirror surfaces. As the curve of the mirror surfaces flattens out the reflex lines tend to converge as seen at the tapering ends of the folds (Plate XXI, fig. 6). Folds in Descemet's membrane are associated with all severe corneal involvements (e.g., especially in interstitial keratitis or after surgical operation). Bowman's membrane, which has not a definite limiting border on its deep side, rarely folds.

Koby pointed out that the reflexes from folds in Descemet's layer must be differentiated from tears. The latter, however, are broader, do not have double reflexes and do not display the parallax displacement characteristic of the folds. These reflexes may be confused with the corneal nerves, but the latter have double or triple ramifications. Vascular remains also may be confused but are easily differentiated when examined in retro-illumination.

INDIRECT ILLUMINATION (PROXIMAL ILLUMINATION OF GRAVES)

This type of illumination combines some features of sclerotic scatter and some of retro-illumination. It is obtained by focusing a

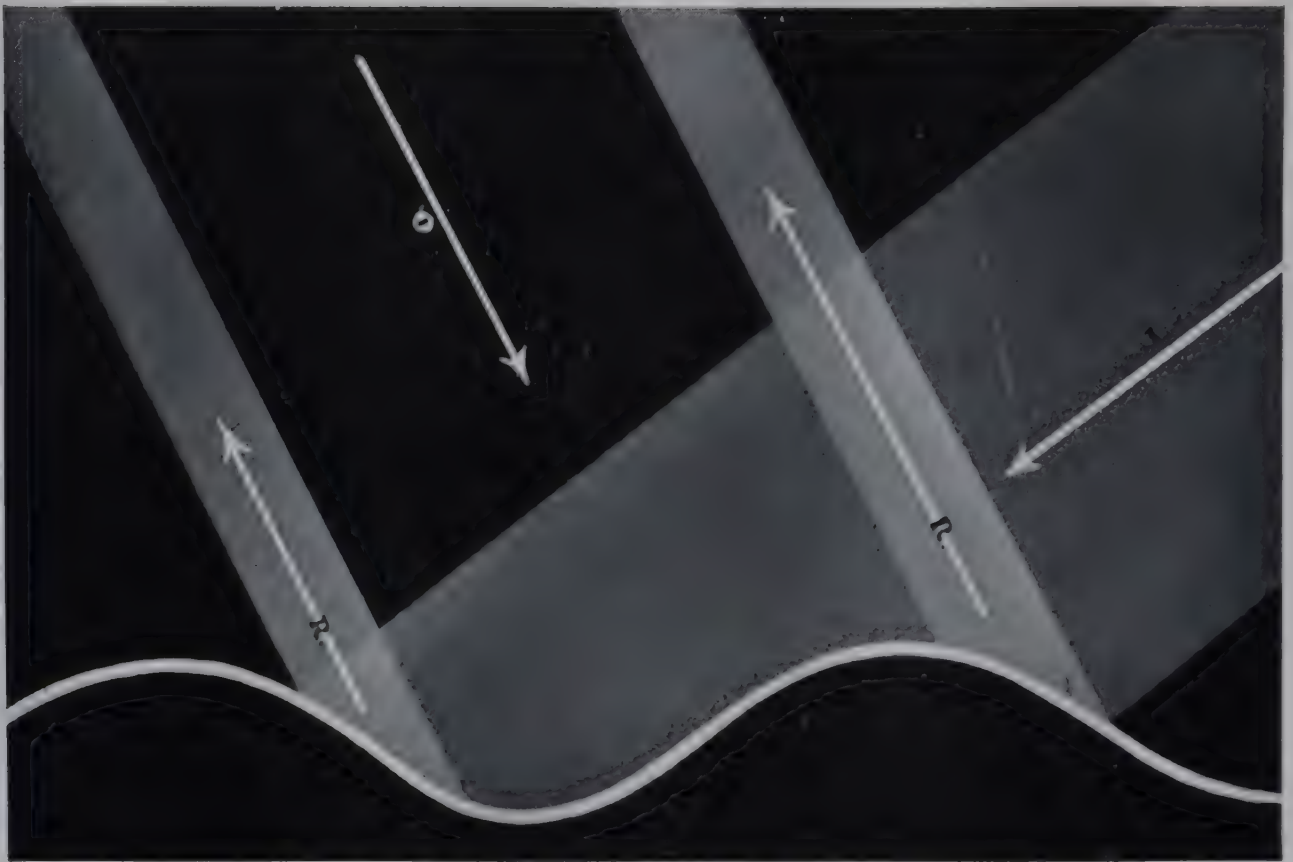


FIG. 97. The production of double reflexes from folds in structureless membranes. The beam of incident light is reflected from the portions of the fold which lie at an angle to the axis of the beam, producing a double line of reflection. *I*, Incident light of beam; *R*, reflected rays; *O*, observer.



FIG. 98. Appearance of folds indicated diagrammatically in Figure 97.

small beam on nontransparent translucent tissues (e.g., sclera, iris, leukoma of cornea) adjacent to the feature under observation, which otherwise cannot be seen by direct focal illumination (Figs. 99, 100).



FIG. 99. Indirect illumination of the iris sphincter (proximal illumination), showing the curved edge of the sphincter to the sides of the bright area illuminated by direct focal light.

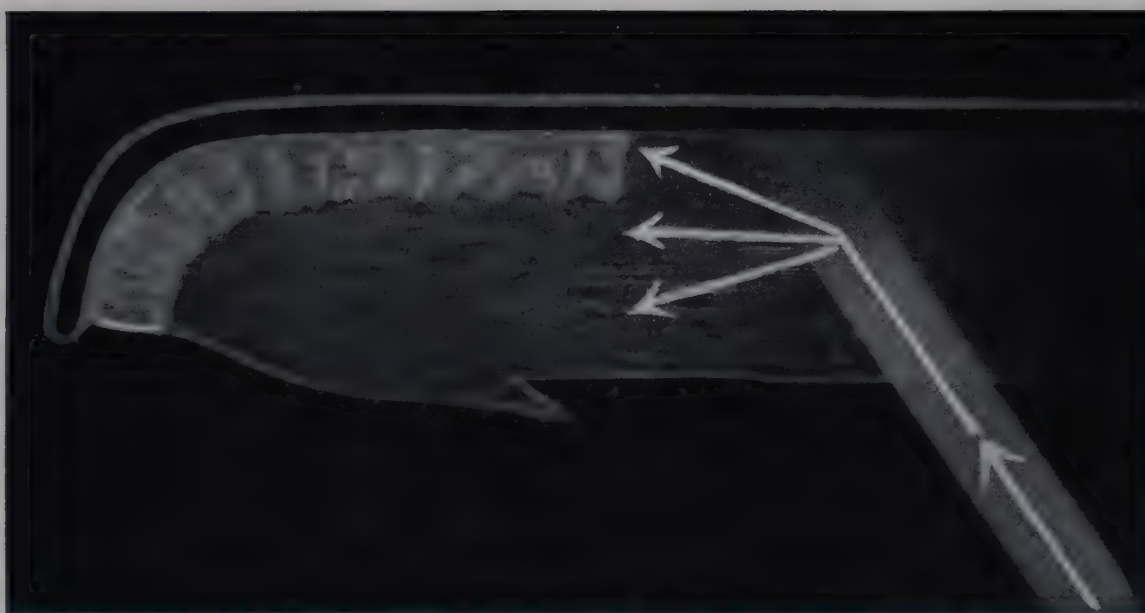


FIG. 100. Diagram of indirect proximal illumination. (Courtesy of T. H. Butler.)

A less readily illuminated tissue, segment, scar or deposit (e.g., deep scleral or iris vessels or hemorrhages in the iris or the sphincter iris itself) stands out darker on a background illuminated by the scat-

tered light. To obtain good results the incident beam should be at a wide angle to the axis of observation. This facilitates internal reflection and scatter of the light. In addition, oscillation of the beam

permits variable illumination, which accentuates certain features perhaps overlooked in fixed illumination. When the microscope is focused back and forth, the plane of the feature under observation is more easily judged.

OSCILLATORY FIELD

Small lateral or vertical movements are made with the beam so that the point under observation is alternately viewed by direct and indirect light. Such oscillatory movements cause sudden alternate lighting up and darkening of fine details. In a sense it seems as though the details themselves are caused to move, and their presence is seen with greater ease than when observed under stationary illumination.

PROJECTION OF SHADOWS

Structures in the anterior layers of the cornea and the lens, when viewed in direct focal illumination, may cast shadows or form so-called "optic reduplications." For example, mucus on the epithelial surface or a foreign body in the superficial corneal layers or a small nebula projects a shadow, which may be seen as a linear dark tapering streak, extending to the posterior face of the optic section (Fig. 101). Pigment on the anterior capsule produces a similar effect in the lens. Vessels, blebs, scars, and innumerable types of structures cast shadows, depending on the manner in which they obstruct, absorb, refract, and scatter light (Fig. 102). In the cornea, owing to these phenomena, such superficial structures, when viewed in the parallelepiped or in optic section, frequently produce "optic re-

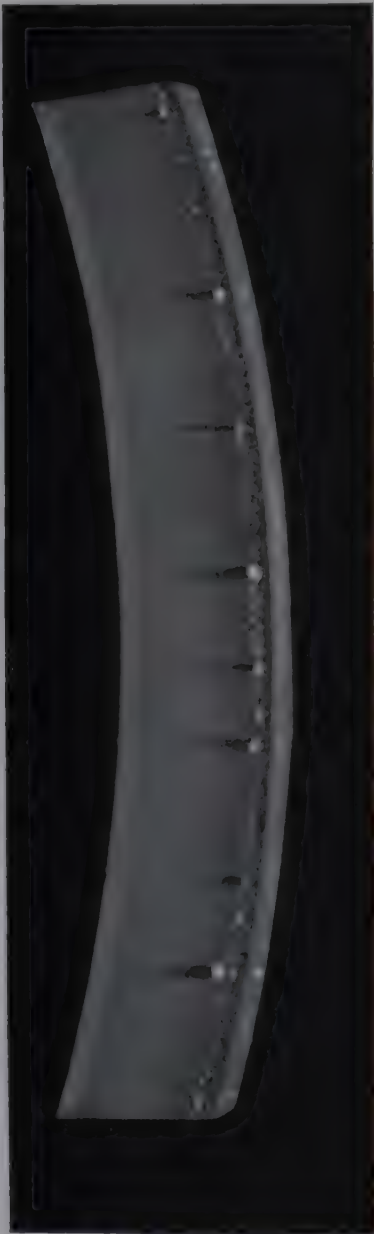


FIG. 101. Projection of shadows in parallelepiped from opacities in Bowman's zone.

duplications" on the posterior face, similar to a shadow picture. In other words, the posterior face acts like a screen, on which an exact replica of the anterior features which obstruct the light is projected



FIG. 102

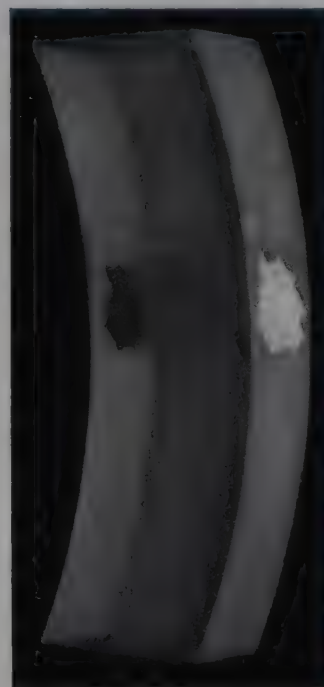


FIG. 103

FIG. 102. The converging action of a large lenslike bulla producing a luminous cone within the optic section.

FIG. 103. Optic reduplication in parallelepiped upon the posterior corneal surface of an obstructive feature situated on the anterior corneal surface.

as a shadow (Fig. 103). These shadows may be mistaken for pathologic changes. Even though they are more transparent than the media in which they lie, such features as vacuoles or blebs in the cornea or lens may project shadows on deeper surface in optic section. This is due to the smooth mirror-like anterior and posterior surfaces of the vacuole which reflect the light more strongly than the surrounding media, producing bright reflexes and diverting the incident light so that a shadow of the vacuole is formed, the apparently transparent vacuole acting as an almost opaque body projecting a shadow (Fig. 104).

If a large homogeneous bulla is observed in direct focal illumination, there are three refractile phenomena that may occur (Koby): (1) If the refractive index of the contents is greater than that of the surrounding media, it acts like a convergent lens, producing a luminous cone bordered by two dark shadowy bands. (2) If the refractive index of the contents is less than that of the media a di-

vergent lens action results. This leads to a projected shadow of the bulla, encircled by a more or less divergent luminous beam. (3) When the refractive index of the body is the same as that of the media, a direct shadow formation occurs (Fig. 105).



FIG. 104

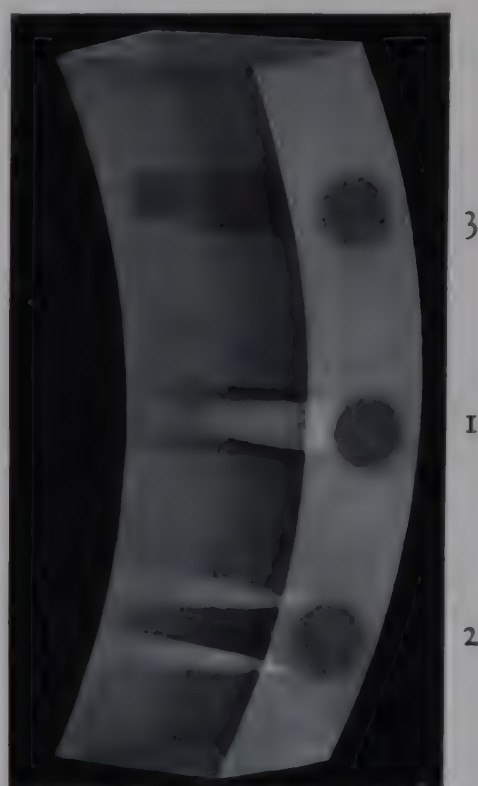


FIG. 105

FIG. 104. Shadow formation in the case of a smooth bulla (contents are of the same refractive index as the surrounding media). Owing to the mirror-like properties of the smooth external and internal surface of the bullae considerable incident light is "used up." Therefore, by refraction less light is transmitted through the bulla and a shadow results.

FIG. 105. Shadows cast by bullae. Refractile phenomena caused by bullae. 1, The index of refraction of the bulla is greater than that of the media causing a convergent lens action. 2, The index of refraction of the bulla is less than that of the surrounding media causing a diverging lens action. 3, The index of refraction of the bulla is the same as the index of refraction of the media. Direct shadow formation occurs. (After Koby.)

The formed replica of such a lesion on the posterior face of the cornea might be mistaken for pathologic change but for the fact that there is always a projected shadow through the opalescent block in front of it. Similarly, subcapsular lenticular vacuoles may produce shadows. Small corneal scars, when viewed in diffuse illumination, cause a refractile disturbance, which may produce a dark shadow on the surface of a lightly colored iris or even on the anterior lens capsule. Shadow streaks caused by artifacts (dust, dirt on the lenses or on the slit blades) in the illuminating system may cause shadows which resemble those associated with pathologic fea-

tures and may be misinterpreted. The artefact shadows move with the motion of the beam, while shadows from pathologic structures are fixed in the tissue.

METHODS OF LOCALIZATION

Biomicroscopy permits exact localization of features under observation. In transparent tissues, such as the cornea or the lens, diagnosis is sometimes entirely dependent on localization, because in certain diseases, lesions such as infiltrates and new vessels seem to have a predilection for specific sites.

Before the development of biomicroscopy, localization depended on stereoscopic judgment and possibly comparative refraction of the observer or, if a monocular loupe or ophthalmoscope were used, on the experience or skill of the examiner in determining the location of the object in relation to structures of known depth. Butler states "that the power to localize is the chief feature of the slit lamp and the student should endeavor to become so proficient that accurate localization is a mere part of observation entirely automatic and intuitive."

Several methods of localization are afforded by biomicroscopy. The most valuable, from a purely clinical standpoint, are those which employ direct focal illumination (the parallelepiped and the optic section) and relative focusing of the microscope.

LOCALIZATION BY DIRECT FOCAL ILLUMINATION (VOGT)

This method is based on the fact that a parallelepiped or optic section of the ocular tissue is formed when the focused beam passes through the relucient ocular media of the cornea or lens. A narrow knifelike beam should be used for exact localization, as it eliminates the interference from the broad surfaces of the blocks (Fig. 106) which is produced by the wider beam. If the beam is directed along the optic axis or perpendicular to the surface of the cornea, only the anterior face of the block is seen; by angulating the incidence of the beam 30 degrees or more, the side walls of the corneal parallelepiped are discerned. This procedure may be compared with the cutting of

a small block of the total thickness of the corneal tissue and turning it to bring the side walls into view.

With the accurate focusing of the wide beam on the cornea a



FIG. 106. Localization of a detail in the corneal optic section. The distance from the anterior surface is represented by the shorter arrow. The longer arrow represents the distance to the posterior surface.

sharply defined relucant block is obtained (Fig. 107). Its superficial face, *abcd*, is curved. This curvature, which is governed by the anatomic shape of the corneal surface, is increased as the angle of incidence of the beam becomes greater. As the beam traverses the cornea, the curvature of the surfaces of the corneal parallelepiped and its apparent thickness vary because of the change in the form of the corneal section. In addition, there is a curved posterior face, *efgh*, representing the posterior surface of the cornea; a proximal lateral face, *aecg*, nearer the observer, which is distinctly seen, as compared with a distal lateral face, *bfdh*, which is viewed through the relucant substance of the block. As this block is viewed stereoscopically with the Czapski microscope, its surfaces and lateral walls are em-

ployed as fixed planes of reference for localization. As the size of the beam is narrowed by diminishing the width of the screw-operated slit aperture, the lateral walls approach each other, thus narrowing the widths of the anterior and posterior surfaces of the block, until a section as thin as 20 microns is obtained. This manipulation enhances the view of the lateral walls, which represents a section through the thickness of the tissue. At this point the corneal parallelepiped with its four surfaces is converted into a single plane, forming a hyperboloid. Vogt terms this an "optic section." Its value lies in enabling the observer to judge structural configuration and localization of pathologic features therein. For example, in observing a superficial corneal scar in the parallelepiped, the impression may be gained that a flattening of the cornea is present (Fig. 238).

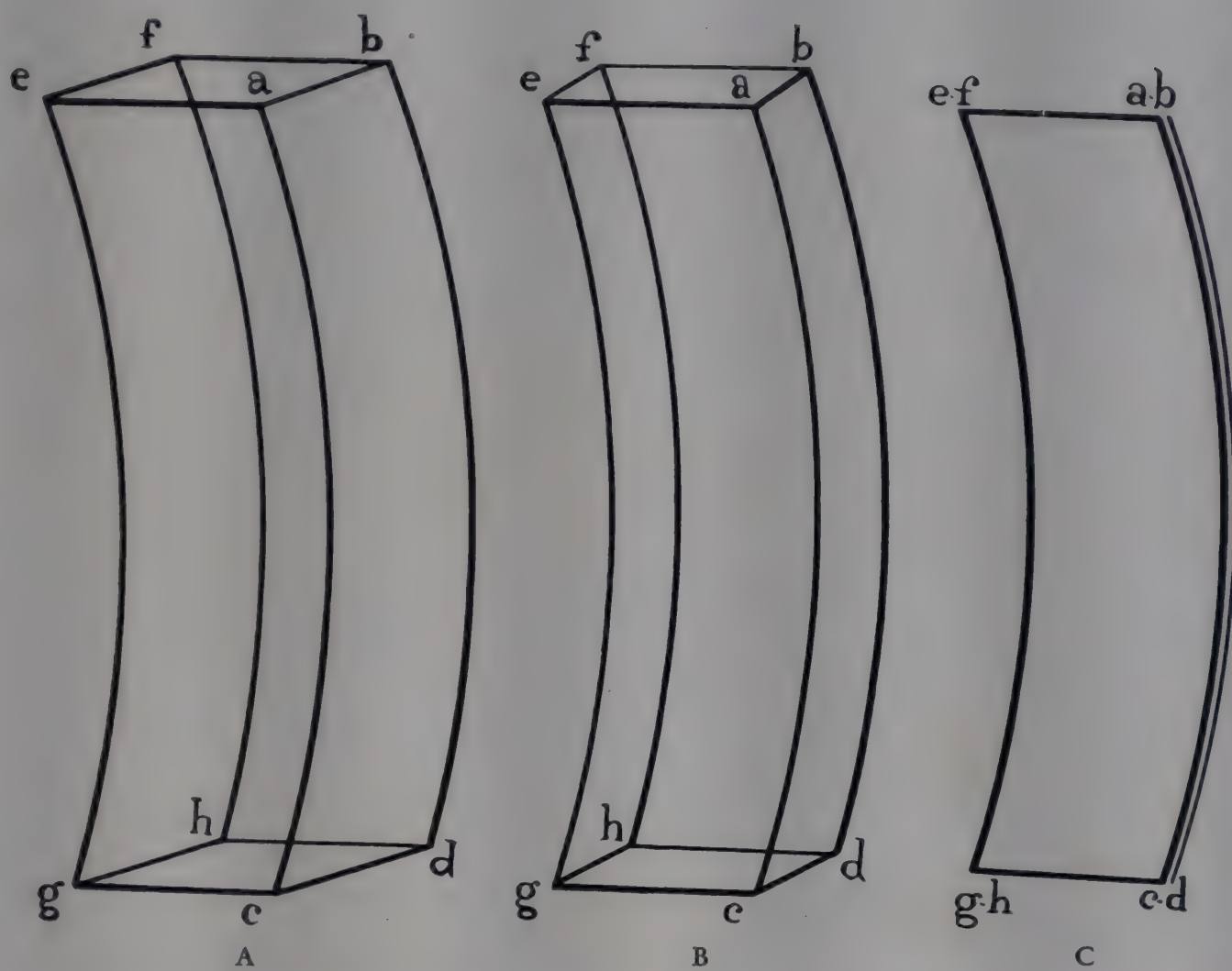


FIG. 107. Diagram showing the effect of narrowing the width of the slit, transforming the corneal parallelepiped into an optic section. A. Corneal parallelepiped. B. A narrower parallelepiped obtained by narrowing the slit diaphragm. C. Optic section obtained by maximum narrowing of the slit diaphragm.

However, when the same region is observed in the optic section, it will be seen that the flattening is illusory, owing to the invisibility of the clear, dark nonresponsive epithelium, which has filled in the defect to maintain the normal surface contour of the cornea. The pre-corneal line is unchanged in curvature and appears to arch over the thickened epithelium in the defect. The optic section is especially useful in demonstrating thinning or thickening, either localized or general, of the corneal thickness.

Minute changes are readily detected because of variations in the refractive index, which produce an increase in relucency as compared with the normal opalescence of the corneal section. Localization of these features can be attained by noting their position stereoscopically in relation to (1) the surfaces of the block or the section, or (2) the level at which they intersect the proximal lateral face of the section.

It should be noted that a structure situated on the anterior or posterior surface of the tissue will appear sharp only in the corresponding face and not in the intermediary layers of the section. Any intermediate pathological feature will lie between the two faces.

For purposes of localization, it is important to focus on the feature within the optic section and accurately to determine the level at which the feature cuts the optic section. One should avoid viewing the object by retro-illumination against the deeper posterior portions of the section because this might cause the observer to localize an object (especially a blood vessel) at a plane deeper than the one in which it actually lies. The relative change in depth of sectional features and those outside the section which apparently lie on it, cannot be represented stereoscopically in a flat drawing. Therefore, Graves has suggested that features which are outside the section but owing to its obliquity seem to lie over it nearer the observer, be termed "extrasectional" to distinguish them from purely sectional features.

When attempting to localize a pathologic feature, it is also important to realize that the deeper portions of the optic section are

seen through the overlying tissue to the observer's side; if this tissue is altered it may obstruct or distort the view of these portions. This does not occur when only the posterior layers are involved. In order to bring about this obstruction, the pathologic area must be of sufficient breadth to cover at least part of the lateral wall of the optic section facing the observer. Graves has termed this "refractile" distortion, "observational," in contrast to "primary" distortion of the deeper part of the optic section itself, resulting from refractive changes by surface irregularities of the lesion through which the optic section is passing (Fig. 108).

Thus, "primary" or real distortion is observed in the optic section when it passes through a raised bulla on the surface of the cornea to one side of the center of the bulla. It appears as a bulging or concavity of the deep part of the section, depending on whether the beam cuts through the bulla on the side away from the observer or through the portion proximal to him (Figs. 109, 110). This is caused by a primary bending or warping of the otherwise flat plane of the posterior surface of the optic section owing to the fact that the bulla acts as a lens. The intersection of this warped optic section with the posterior surface of the cornea produces an apparent distortion of the posterior boundary of the section.

In optic section, the presence of reduplication lines on the anterior surfaces of the cornea and of the lens aids in anterior localization.



FIG. 108. Secondary (or observational) distortion in the case of corneal bulla. Illustrating the distortion of the posterior edge of the optic section when viewed through the extra-sectional part of the beam.

In the deeper parts of the lens, further assistance is afforded by the zones of discontinuity, resulting from its internal architecture.* A method of estimating the millimetric distance of pathologic fea-

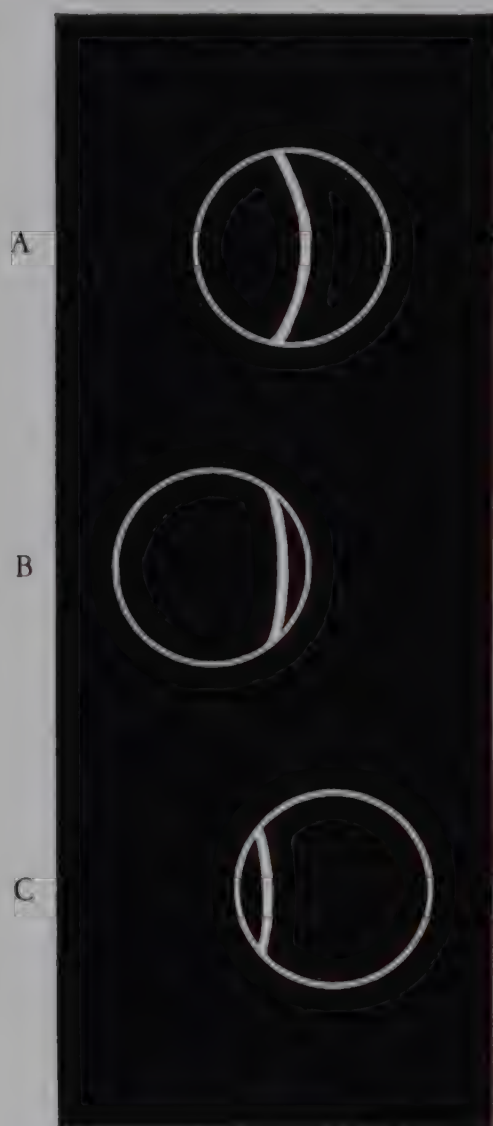


FIG. 109

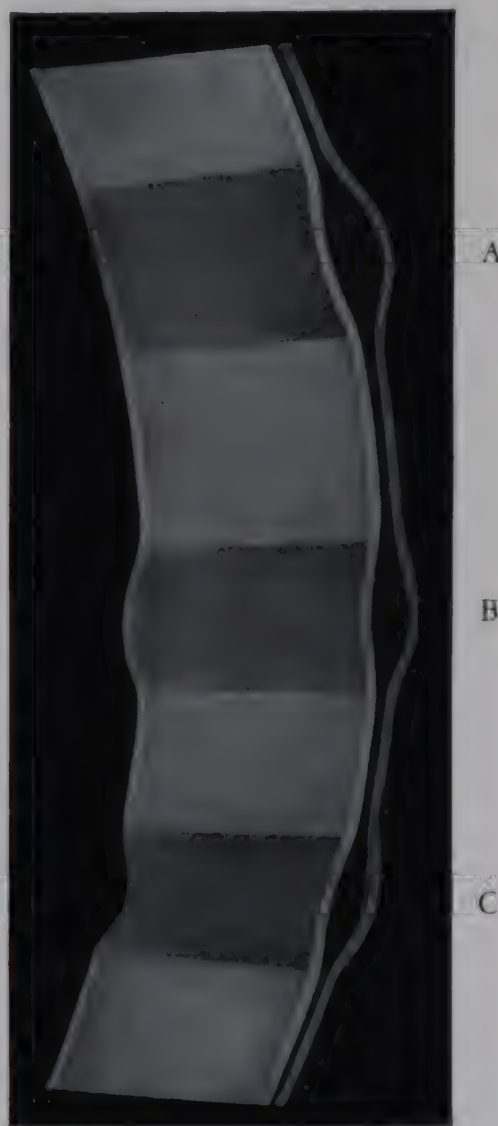


FIG. 110

FIG. 109. The positions of optic section as it traverses circular bullae. A. Section passing through the center of a bulla; B, section passing through the portion distal to observer, and C, through the portion proximal to observer.

FIG. 110. Resulting primary distortion of the optic section in the above conditions shown in Figure 109. A, No distortion of the plane of the optic section; B, convex distortion resulting in an apparent convexity of the posterior edge; C, concave distortion resulting in an apparent concavity of the posterior edge.

tures in the cornea has been suggested by Vogt. He assumed that the central thickness of the cornea is from 0.8 to 1.0 mm. With a magnification of $20\times$, the section would have an apparent breadth of 16 mm., which must be halved because of the angle of the section. This gives an actual line of 8 mm. for purposes of measurement.

* Localization in the lens is considered in more detail in Volume II.

This line is mentally divided into millimeter segments (the actual length of which is 0.1 mm.). Hence, if a pathologic feature is 2 mm. from the corneal surface, as seen in the image, it is actually one fifth of a millimeter from the anterior corneal surface. Koby found that the corneal thickness is less than is generally believed, being 0.6 mm., and not 0.8 mm., as Vogt estimated. The error in this method depends on the personal equation of the observer. In addition, variations in corneal thickness interpose disturbing alterations, which may lead to gross inaccuracy especially in the presence of other pathologic processes.

LOCALIZATION BY FOCUSING THE MICROSCOPE

The Czapski microscope with its stereoscopic principle offers marked improvement over the small monocular loupe in evaluating the relative depths of details. With it, the observer soon learns automatically to localize by employing reference points of known depth.

The microscope is focused along the optic axis with direct focal illumination on points of known depth, such as the anterior and posterior surfaces of the cornea, the surface of the iris, the anterior and posterior lens capsule. The object, the depth of which is being determined, is then illuminated by reflected light (retro-illumination or scatter). If the object is in another plane than that of the known point in sharp focus, it appears blurred, and displacement of the microscope is required to bring it into sharp focus (Fig. 112). The amount of displacement of the microscope that is necessary is a measure of the depth of the object from the point originally focused. For example, to determine the depth of an opacity in the anterior portions of the lens, the microscope is first focused on the anterior lens capsule, employing direct focal illumination. An opacity in the anterior adult nucleus appears somewhat blurred, and a forward displacement of the microscope is required to bring it into sharp

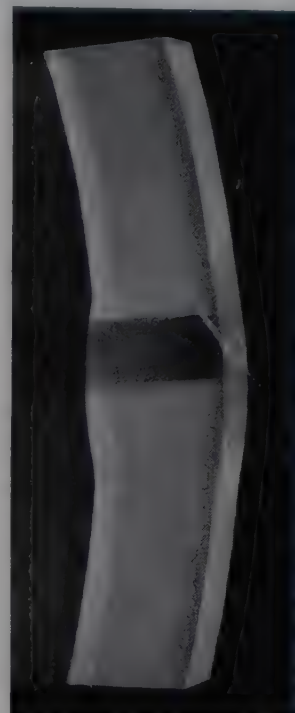


FIG. 111. Herpetic lesion (regressive stage) showing apparent distortion of the posterior corneal surface.

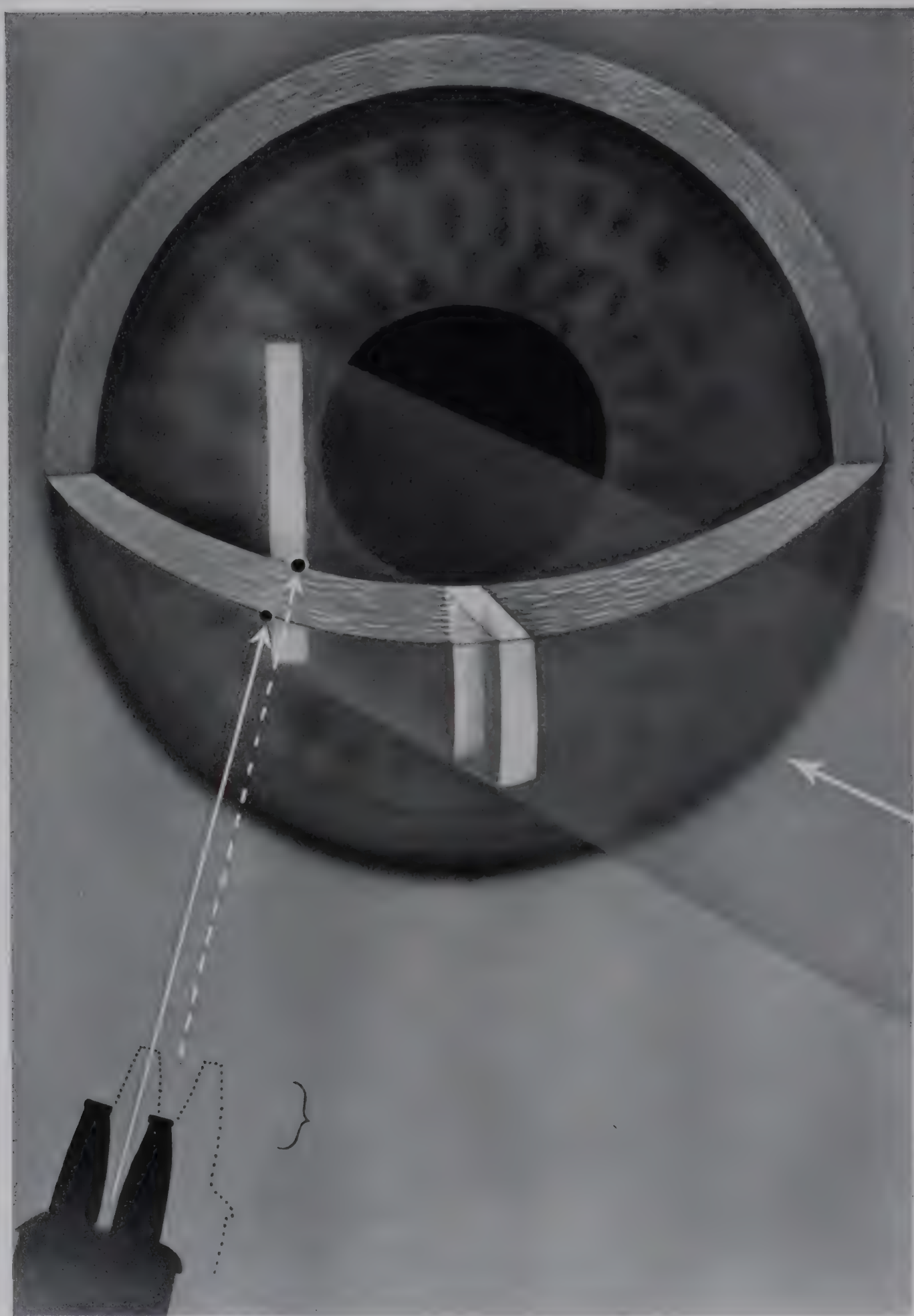


FIG. 112. Localization by microscope displacement in retro-illumination. Dotted line indicates focus on detail (keratic precipitate) on posterior corneal surface. Solid line indicates microscope displaced backward to focus on detail on anterior surface. Width of the bracket indicates the amount of displacement of the microscope.

focus. For points situated still deeper, nearer the posterior surface of the lens, the posterior lens capsule serves better as the reference point. Droplets of edema of the corneal epithelium (bedewing) are not easily distinguished in direct focal illumination; retro-illumination is required to determine their identity, especially when other corneal alterations are present. Since the droplets lie in one plane, their location can be determined by focusing the microscope. Features on the posterior surface of the cornea, such as a keratitic precipitate, are brought into focus. The epithelial droplets are then blurred and in order to focus them sharply it is necessary to displace the microscope backward, thus indicating their superficial position with relation to the keratitic precipitates (Plate XXV, figs. 3, 4). Naturally, in these processes the stereoscopic depth perception properties of the binocular microscope, which stimulate the psychological judgment of the relative position of an object in space, are an aid. It is imperative that good fixation and the patient's complete co-operation be obtained.

The Ulbrich Micrometer. The foregoing method is applicable to quantitative mensuration if a precision instrument, such as an Ulbrich micrometer, is used. However, the margin of error in such estimations is fairly high and, therefore, these measurements are not easily applicable to ordinary clinic practice. The Ulbrich drum, which is a micrometer scale mounted on a metal cylindrical drum (Fig. 29), measures the displacement of the microscope as it is moved from one known point of focus to a second known point. Its chief practical value lies in the fact that it affords a means of measuring the depth of the anterior chamber and the lens. The thinness of the cornea makes its mensuration difficult.

The drum is affixed to the left pinion of the microscope-focusing mechanism. The scale is graduated in 0.1 mm., and records the anterior displacement of the microscope along its axis. In determining the depth of the anterior chamber, the null point of the drum setting is obtained by focusing the microscope sharply on the vertex of the posterior corneal surface. Butler recommends using for this purpose

the endothelial cells, which are seen in the zone of specular reflection on the posterior surface of the vertex of the cornea. By operating the right hand milled screw of the focusing mechanism, the microscope is moved anteriorly toward the eye under examination, until the lens shagreen is clearly defined — or the pupillary margin of the iris, if the eye is aphakic. As high a magnification as possible should be used, since the shorter range of focus which is associated with the higher magnification ensures more exact focusing. It is necessary to illuminate the eye with light of high intensity.

The measurements obtained with the Ulbrich drum are virtual owing to the refractive error introduced by the cornea-aqueous refractive system. Without a corrective factor, Butler determined the chamber depth to be 3 mm. with this method. Hartinger¹⁴⁵ gave a corrective formula for obtaining absolute values from the virtual measurements which are made with the microscope displacement method. A corneal thickness of 0.5 mm. is employed in this calculation. Gradle and Sugar,¹²⁴ employing Hartinger's corrective formula, found that in 100 normal eyes of patients over 55 years of age, the average chamber depth was 2.10 mm., measured to the anterior surface of the pupil border of 3 mm. pupils. The true corneal thickness may be measured in a like manner. With direct focal illumination the corneal microscope is focused first on the vertex of the anterior surface of the cornea and then on the vertex of the posterior surface. The displacement of the microscope, as shown by the drum micrometer, gives the apparent thickness of the cornea from which the true thickness must also be calculated. To measure the lens depth, it is necessary to use the narrow beam and to focus both the microscope and the beam on the anterior capsule and subsequently on the posterior capsule.

Ocular (Eye Piece) Micrometer. The area or depth may be measured with ocular micrometers.* Several types are available. An 0.1 mm. graduated metric scale may be used. In order to obtain accurate measurements the true magnification of the optical system

* One of the ordinary oculars is removed and is replaced by an ocular micrometer having a specially etched grid on the *lens* surface.

of the microscope must be determined. This is found by calibrating the ocular micrometer with a stage micrometer. When in use, the number of 0.1 mm. divisions which are covered by the image of the object being measured is counted. This number divided by the magnification gives the true measurement in millimeters. The ocular micrometer may also be used for depth measurements and the approximate calculation of corneal or lens thickness (Vogt). To measure the thickness of the cornea the number of divisions of the ocular micrometer which cover the proximal border of the corneal parallelepiped are determined. This is an apparent projection of the corneal thickness observed perspectively. In order to calculate an absolute figure, the angle between the axis of the microscope and the axis of illumination must be determined. This may be measured with a goniometer. Trigonometric calculation gives an apparent thickness, which, when corrected for refraction, yields the actual corneal thickness. These methods are of significance in experimental studies but are of only theoretical interest to the clinician.

LOCALIZATION BY THE ZONES OF SPECULAR REFLECTION

The fact that the major zones of specular reflection occur at certain levels (surfaces) in the cornea and lens assists in obtaining a fixed point as a plane of reference. Pathological features situated behind the zone (i.e., in the anterior chamber) obstruct none of the details seen in the zone of specular reflection, whereas those in front (corneal) are obstructive. With this method of localization it is possible to determine whether a change lies in front of, in, or behind the endothelium. For instance, the vascular remains of interstitial keratitis are seen as dark lines directly in front of the endothelial mosaic, whereas in endothelial dystrophies the changes produce disturbances within it.

LOCALIZATION BY PROJECTED SHADOWS

Practically all features seen in the transparent ocular media (cornea or lens), whether situated on their anterior surfaces or within these structures themselves, cast shadows posteriorly. These

shadows are projected to the posterior surface and tend to taper off. The length of the projected shadow measures the distance between the observed feature and the posterior surface of the structure in which it lies. Koby¹⁷² elaborated on the principles of this method and also suggested the use of the catoptric image of the intra bulb filament for purposes of localization.

LOCALIZATION BY PARALLACTIC DISPLACEMENT

A further use of "visual palpation" was suggested by Koeppe. He advises creating an oscillatory motion in the beam, thus producing a parallax displacement of objects with relation to each other.

PROCEDURE FOR EFFICIENT BIOMICROSCOPY OF THE EYE

A general survey of the eye is made in daylight or by oblique illumination with the aid of a condensing lens, employing a loupe for magnification; an ophthalmoscopic examination is made, and then the biomicroscopic examination is conducted as follows:

(1) *Sclerotic scatter (under low power)*: By focusing the beam on the temporal sclerolimbic junction, the most delicate corneal opacities can be revealed. (2) *Retro-illumination*: Without changing the focus of the microscope, this is obtained by displacing and focusing the beam on the iris, the pupil, or both, if the lens is opaque. *Direct or indirect retro-illumination* is produced either by varying the direction of the beam or that of the axis of observation. (3) *Direct focal illumination*: both the wide and the narrow beam are employed to produce the corneal parallelepiped and optic section; localization of all features should be emphasized. (4) The *zones of specular reflection* from the anterior and posterior corneal surfaces are then scrutinized. (5) The beam is again focused on the iris and the microscope is used to detect the presence of aqueous turbidity in the anterior chamber, as exhibited by the flare (Tyndall) phenomenon. (6) Following this, the iris is examined in direct focal illumination and by proximal illumination. (7) The beam is then directed into the pupil and the iris transilluminated, if possible. (8) With pupil dilated, if feasible, the lens is examined in direct focal

illumination and specular reflection by focusing the beam on the anterior lens capsule. It is impossible to observe the entire thickness of the lens with a single focus of the microscope or beam. Students are often confused by composite illustrations depicting the beam as apparently in focus throughout its thickness. When the anterior portions of the lens are in focus, the posterior portions of the lens are illuminated by the divergent postfocal part of the rays. It is necessary, then, when examining the posterior portions of the lens, to focus the beam (convergent rays) deeper on the posterior lens capsule and at the same time correspondingly to advance the focus of the microscope. *In order properly to observe the architecture of the lens, it is necessary to use a narrow beam and a dilated pupil.*

(9) The zones of specular reflection from the posterior lens capsule (shagreen) may now be inspected. The anterior portions of the lens may be retro-illuminated by the reflected light from the mirror reflex of the posterior lens capsule. (10) Continuing to advance the focus of the microscope, the portion of the beam focused in the vitreous is now examined. Since a considerable part of the light intensity is "used up" in its passage through the cornea and lens, only the anterior third of the vitreous can be seen well. However, overloading the lamp or use of an arc source improves the illumination. Because of limitations in narrowing the angle between illumination and observation and in the dimensions of the pupil, the slit beam should be projected from the nasal as well as the temporal side to enable the observer to examine as large an area as possible.

Chapter Four

THE NORMAL CONJUNCTIVA

HISTOLOGY

THE conjunctiva is the mucosal lining of the inner surface of the eyelids. In the depths of the cul-de-sac it is reflected over the anterior portion of the ocular globe as far as the sclerocorneal limbus. At the eyelid margin just posterior to the gray line, the cutaneous epidermis of the eyelid changes gradually into the conjunctival mucosa proper. The conjunctiva is applied firmly and smoothly to the internal surfaces of the tarsi, appearing as a reddish, shiny surface of a nonuniform color. The inner and outer angles of the conjunctiva, where the vessels enter, is deeper red in color. Through the semitransparent tarsal conjunctiva the parallel yellowish white lines of the meibomian glands are visible running vertically.

As the conjunctiva approaches the cul-de-sac, its attachment to the deeper layers loosens and it is thrown into folds. Owing to increased vascularity the color of the cul-de-sac is a deeper red than that of the conjunctiva overlying the tarsus. From the cul-de-sac the conjunctiva is reflected over the globe as a pellucid membrane, permitting a view of the sclera. Its attachment to the sclera is lax, allowing gliding movements. As it approaches the limbus it becomes more firmly attached to the sclera and gradually fuses with the corneal surface. At the internal angle, the conjunctiva forms a semilunar fold, appearing as a transparent film ultimately losing itself on the caruncle, and fusing with the modified skin which covers this structure.

A review of the main histologic features of this tissue will aid us in the interpretation of the biomicroscope picture. The con-

conjunctiva (like other mucous membranes) consists of two layers: (1) superficial (epithelial) and (2) subepithelial.

$$\text{Conjunctiva} \begin{cases} \text{Epithelium} \\ \text{Subepithelium} \end{cases} \begin{cases} \text{Adenoid tissue} \\ \text{Fibrous tissue} \end{cases}$$

The *epithelium* of the tarsal conjunction, which is of the stratified type, is composed of a layer of superficial cylindrical cells, superim-

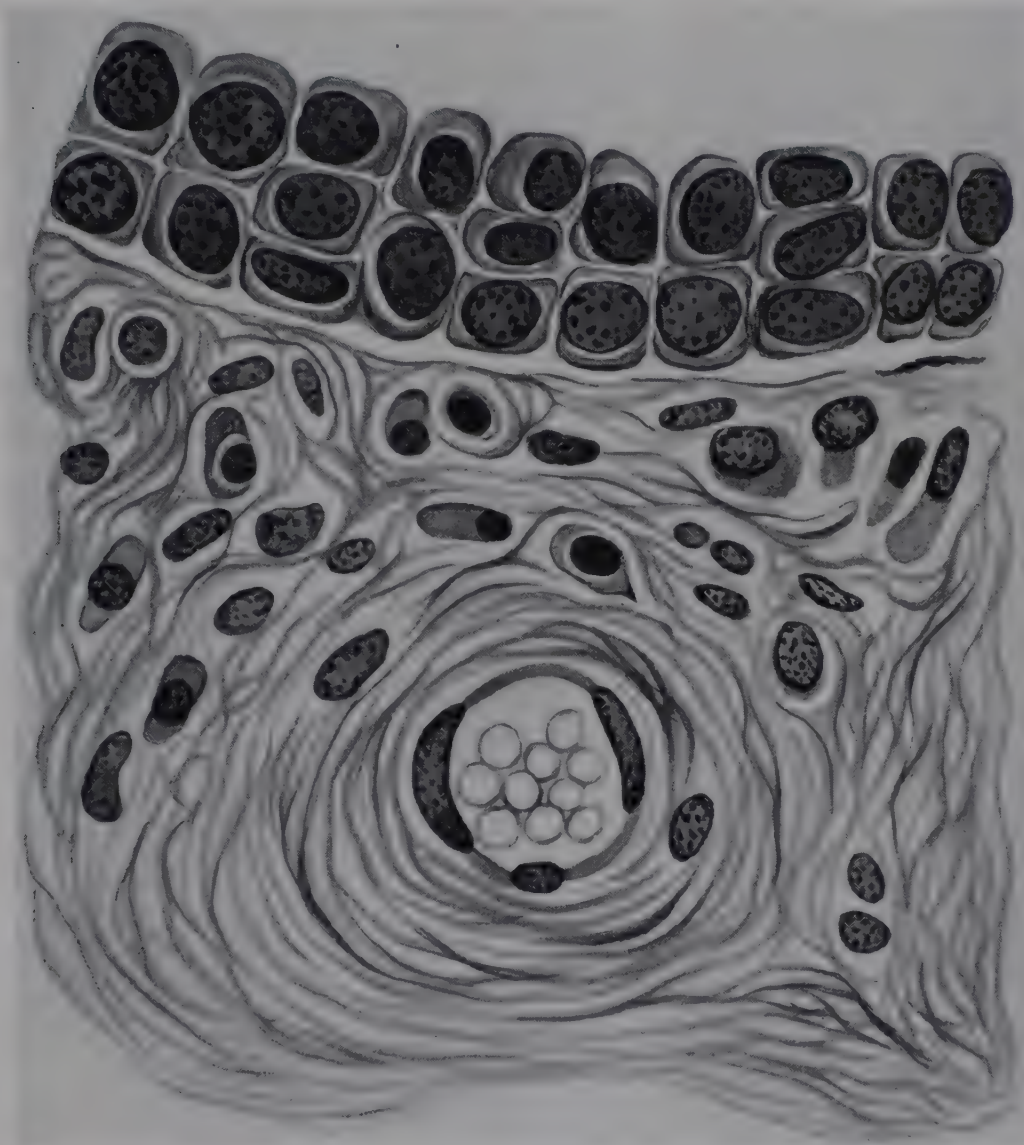


FIG. 113. Histologic section through the bulbar conjunctiva. (Semidiagrammatic.)

posed on a single layer of flatter cells, which in turn rest on a basal membrane. In the region of the cul-de-sac (Fig. 113), the epithelium is composed of three layers of round or polygonal cells. In this form it continues over the bulb up to the limbal region, where it changes into a stratified squamous type. At the limbus it gradually metamorphoses into the corneal epithelium.

The adenoid part of the *subepithelial layer* consists of a loose connective tissue network, separated from the epithelium by a transparent basal membrane which, when it reaches the cornea, merges with the membrane of Bowman. This layer contains the superficial vascular network and gradually changes into the deeper fibrous layer, in which are found elastic tissue, deep blood vessels, nerves, lymphatics, and glands. The fibrous layer is absent in the tarsal conjunctiva; it begins at the upper border of the tarsus to form the deepest zone of the remaining conjunctiva. It is so intimately connected with the episclera that it is thought by some to belong to it.

BLOOD SUPPLY

The blood supply of the conjunctiva is derived from palpebral branches of the nasal and lacrimal arteries of the eyelids and from the anterior ciliary and episcleral vessels, the two systems anastomosing freely.

Tarsal Vessels. Examination of the tarsal conjunctiva with the eyelid everted shows a system of anastomosing vessels which may be divided into three zones (Plate I, fig. 1): (1) short vessels which appear near the margin of the eyelids; (2) long vessels emanating from the edge of the tarsus; (3) anastomotic area between zones 1 and 2.

Bulbar Vessels. The bulbar conjunctiva has a superficial vascular network which may be distinguished from the deeper episcleral vessels (Plate I, fig. 2). It is formed by the anastomosing branches of the medium-sized and smaller vessels derived from ramifications of the larger superficial ones. Frequently, a larger superficial vessel makes its way almost to the limbus and then suddenly changes its course to anastomose with the deeper network. The visibility of the deep network may depend in part on the transparency of the conjunctival layers. The gliding movements of the conjunctiva cause a corresponding shifting of the superficial network; the deeper stationary network of the episclera acts as a background. These two systems anastomose through vessels which traverse the potential pre-formed lymph space (which contains loose connective tissue) be-

tween the conjunctiva and the episcleral tissue. This space may be made visible by the use of lymphagogues (dionine).

The vascular system of the bulbar conjunctiva may also be divided into three zones: (1) the immediate area of the fornix; (2) the immediate area of the limbus; and (3) an intermediary zone. In the first two zones efferent as well as afferent vessels are present, but it may be difficult to distinguish between them. The direction of the blood current in the intermediary anastomosing zone may change if temporary alteration in pressure is exerted by the other two zones, much as a bubble of water shifts if the water level is tilted.

Limbal Vessels. In the region of the limbus, there is a series of vascular arcades derived from the superficial pericorneal plexuses (Fig. 114). These arcades form a complex zone of anastomosing loops. The final branches of the arcades lie over the superficial limbal spur and bend back abruptly to form venules which drain into the corresponding venous plexuses. In some instances, owing to the absence of blood, the terminal loops appear as whitish lines; but if they are subjected to mechanical irritation or massage one can see them fill with blood after a short interval. At the apex of these terminal loops the direction of the current changes and the width of the vessel increases, thus assuming an apparent venous function. Rollin describes an even finer system of arcades in the limbal region, which he states can be seen with difficulty, even with the biomicroscope. He believes that these vessels are venous in nature and that they first separate and then merge, forming a miniature "vena portae," and thereby increasing corneal nutrition.

K. W. Ascher³³³ states: "Biomicroscopically visible vessels were observed in the limbal and paralimbal region of living human eyes, containing highly diluted blood or a clear colorless fluid and emptying into the conjunctival and subconjunctival venous meshwork. I would suggest that these vessels be called aqueous veins, and define them as biomicroscopically visible pathways of blood vessel-like appearance, containing diluted blood or clear fluid, and intercalated,

PLATE I

FIG. 1. Vascular architecture of the tarsal conjunctiva (everted eyelid), demonstrating zones of short vessels, long vessels, and intermediate areas of anastomosis.

FIG. 2. Direct focal illumination (fairly wide beam) of normal bulbar conjunctiva showing vascularization (high power).

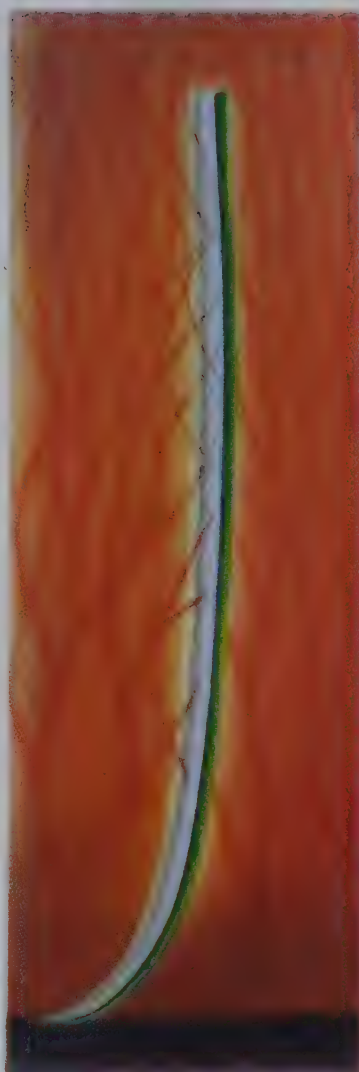
FIG. 3. Optic section through the palpebral (tarsal) conjunctiva showing different layers (low power).

FIG. 4. Optic section through palpebral (tarsal) conjunctiva (high power). (See Fig. 116.)

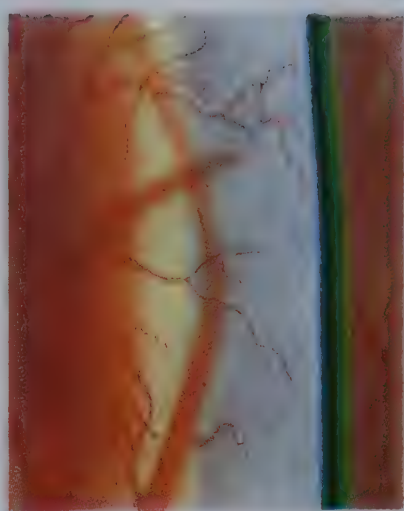
FIG. 5. Optic section at limbus in palisade zone.



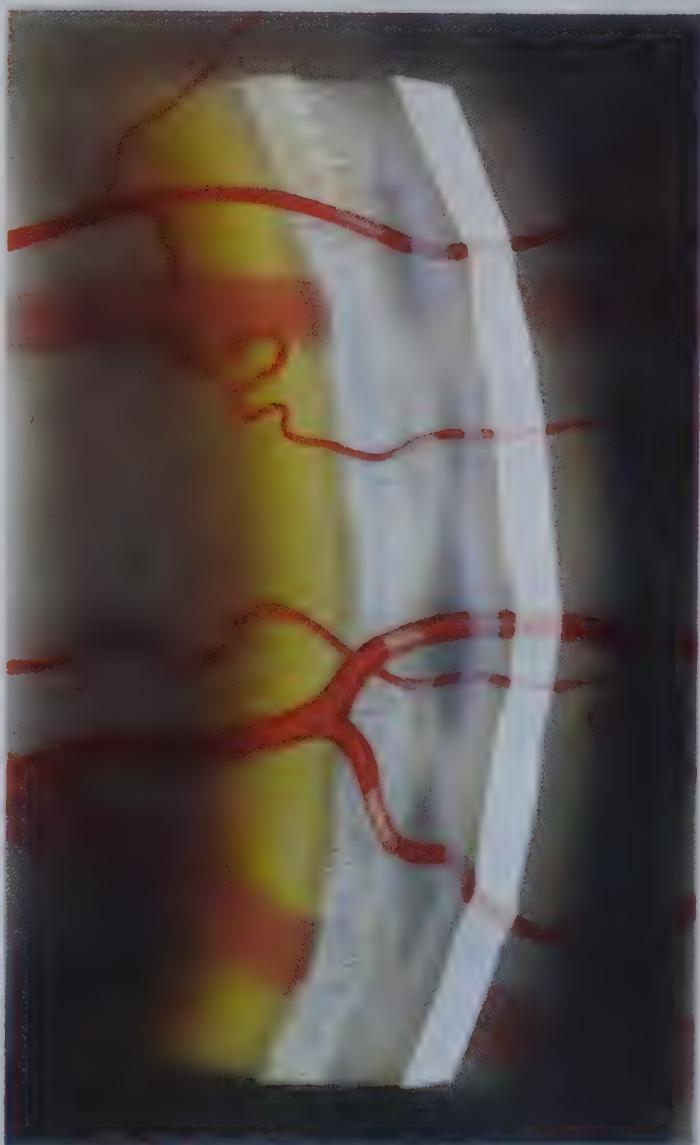
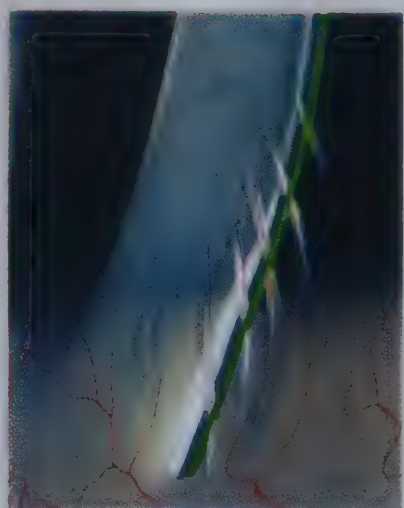
1



2



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probably via Schlemm's canal, between intraocular fluid and conjunctival and subconjunctival veins.

"Aqueous veins are recognized: (1) by their more or less char-

R.I.

D.F.I.

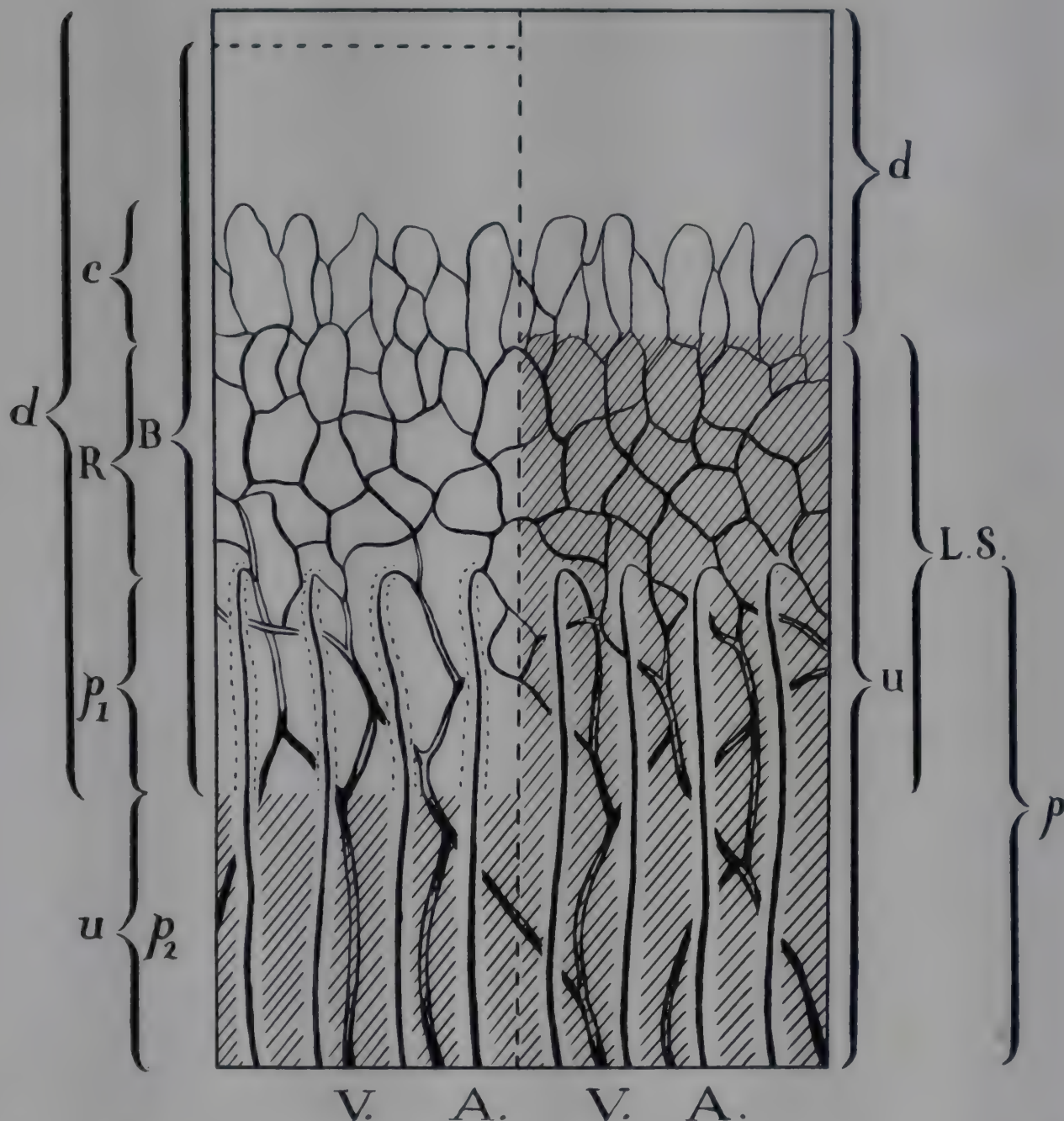


FIG. 114. Diagram of normal epibulbar vascular system at the limbus by retro-illumination (R.I.) and by direct focal illumination (D.F.I.). *d*, Clear portion of cornea; *u*, unclear portion of cornea; *a*, artery; *v*, vein; *p*, *p*₁ and *p*₂, palisades zone; *B*, physiologic edema zone; *c*, terminal vessels of arcades; *L.S.*, limbal spur; *r*, area between terminal vessels and end of palisade zone. (After Vogt.) (See also Plate II, figs. 3 and 4.)

acteristic origin in or near the limbus corneae, or out of an emissarium sclerae; (2) by lack of blood corpuscles as compared to ordinary conjunctival and subconjunctival vessels and, sometimes, by a typical stratification; (3) by their characteristic emptying into

recipient vessels; and (4) by significant phenomena produced by compression of their recipient vessels" (Fig. 115).

The question arises whether such vessels actually contain a special

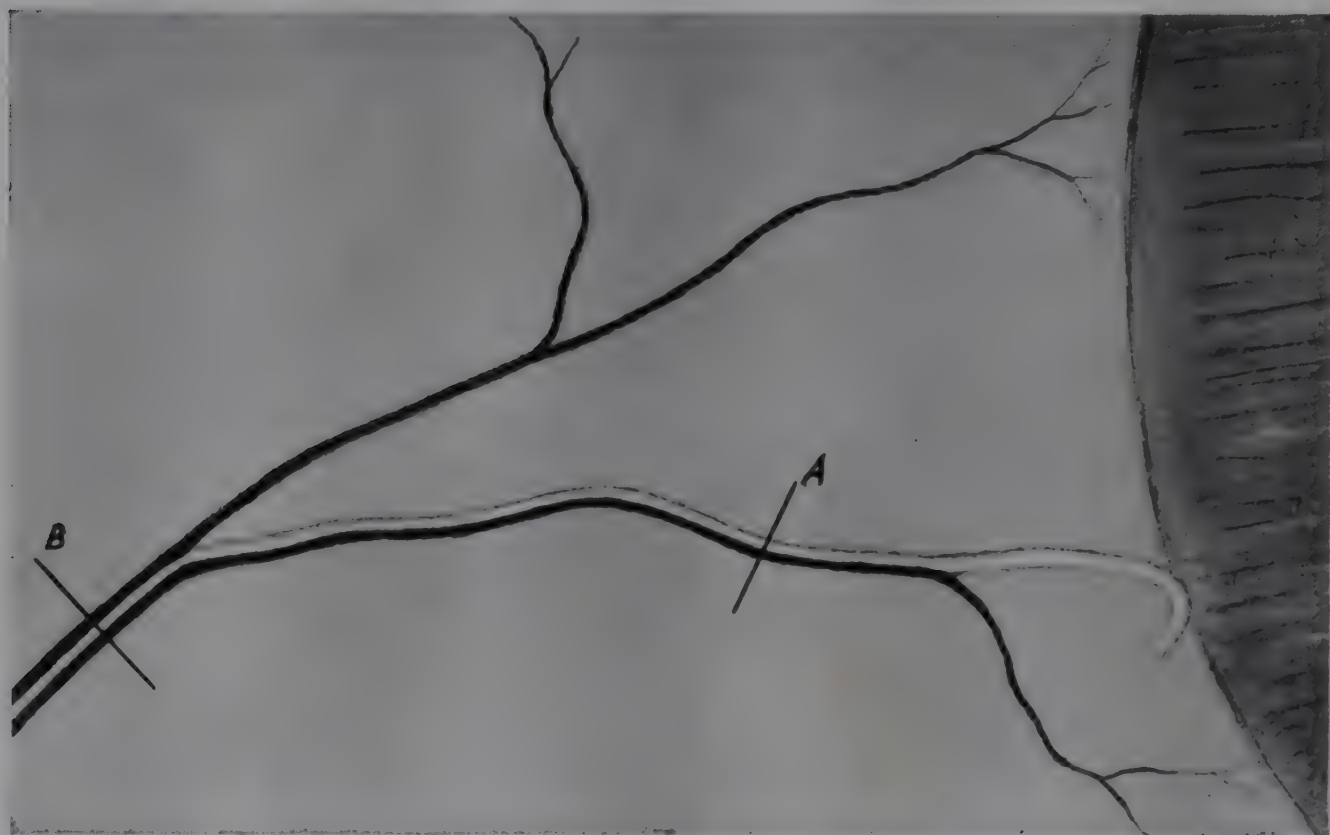


FIG. 115. Origin and source of a typical aqueous vein, in the left eye of a 48-year-old white male. From the lower nasal limbus an aqueous vein starts with an archlike origin and empties into a conjunctival vein coming from the lower bulbar conjunctiva. Below the junction two separate strata are visible, A, the clearer one, being the continuation of the aqueous vein. After joining one more vein the vessel becomes composed of two peripheral red and one pale central layer, B (*Am. J. Opth.* 25:31, 1942.)

clear fluid or whether, owing to their narrow lumens, they simply do not permit the passage of blood corpuscles. Lowenstein²⁰⁰ says: "If these capillaries are to function they must widen, as a blood corpuscle has a diameter of 7 microns. . . . I am able to prove by means of the slit lamp that such a vessel system exists in the conjunctiva. To control the minute conjunctival vessels a drop of ethyl morphine hydrochloride is administered. The small vessels under observation dilate widely, and a great mass of new vessels appear, which were apparently empty before." It is not unlikely that this meshwork is part of a reserve vessel system. However, if Ascher's contentions are correct it would follow, as he states, that, "the detection of aqueous veins has given obvious confirmation to the fact of a continuous fluid elimination from the intact human eye,

according to Leber's theory. Continuous formation of intra-ocular fluid is postulated to balance at least the amount of fluid leaving the eye by the aqueous veins."

CIRCULATION OF THE CONJUNCTIVAL VESSELS

With the magnification utilized in the biomicroscope, it is impossible to visualize individual red blood cells in the vessels; but a granular current may be observed in the smallest branches. The direction of the current, which at times appears to change, does not assist in differentiating between arteries and veins. A more definite distinction may be made by studying the color of the vessels. In the larger vessels no granular circulation is apparent owing to the solidity of the blood column. It is only when fragmentation of the blood column is present that the granular movement is discernible. The granular movement of the corpuscular elements in small vessels of the conjunctiva is best seen by direct illumination with high power. This movement may disappear if the vessel suddenly dilates. Pulsations which are apparently independent of the arterial pulse may occur in these vessels.

Coccius, in 1852, was perhaps the first to observe the blood circulation in the conjunctival vessels. The circulation in the capillaries of the conjunctiva was also seen and described by von Schleich in 1902. Bajardi⁹ describes typical findings in the conjunctival capillaries in cases of arteriosclerosis, syphilis, gout, diabetes, and nephritis, as well as in cataract and synchysis scintillans. Müller recorded similar findings in the skin, occurring in the same diseases. According to Zeller³⁴¹ distention of the vessels is typical in arteriosclerosis; this distention causes the vessels to assume a tortuous course. Rollin²⁵³ stated that in cases of hypertension accompanied by renal insufficiency, there is a tendency toward the formation of aneurismal distentions of the peripheral vessels, whereas without renal involvement these changes are not found (Fig. 116). In syphilis, fusiform or sacciform dilatation of the vessels is observed. In diabetes the venous part of the loop as well as the intermediary part is enlarged. Moreover, if there is a small amount of edema, capillaroscopy pre-

sents many peculiar features. According to Ruedemann²⁵⁷ "thrombi and emboli are present more commonly than had been suspected. Decrease of the capillary bed in the conjunctiva has been coincident



FIG. 116. Conjunctival capillaroscopy in arteriosclerosis, nephritis, and leukemia. 1-12, arteriosclerosis; 13-21, nephritis; 23-24, leukemia. (After Miller.)

with the similar change shown in the histamin flare test. The vessels can be measured and counted and, to date, the patients have experienced no deleterious effects from the illumination [required for this observation]. . . . Although time-consuming, it seems likely that when normal standards have been established, the study will reveal sufficient information to warrant investigation of the conjunctival capillaries in all cases in which blood or vascular disease is suspected."

EFFECTS OF CERTAIN DRUGS ON CONJUNCTIVAL CIRCULATION

Vasoconstrictors (*Epinephrine, Posterior Pituitary Extract, Cocaine, Eucatropine*). Epinephrine (adrenalin) has the most effective vasoconstrictor action. Soon after the instillation of two drops of 1:1000 solution of epinephrine, the veins become narrow and the current is slowed and at times ceases; the arteries blanch, disappear, and give the conjunctiva a porcelain-like appearance.

Vasodilators (Dionine, Physostigmine, Pilocarpine, Histamine). After a drop or two of a 2 per cent solution of dionine (ethylmorphine hydrochloride), venous and arterial vessels dilate to a maximum degree; the blood flow is slowed and within a short time may cease. The conjunctiva is raised because of increased fluid in the subconjunctival space. Pilocarpine likewise acts as a vasodilator, but to a less degree; it lacks the lymphagogue properties of dionine.

When a solution of histamine is instilled in the conjunctival sac, it primarily dilates the capillaries and to some extent contracts the arterioles, and as a result produces a marked chemosis. However, in a congested eye, histamine does not further increase capillary dilatation but rather has a constricting action on the arterioles. In each case, the action of these drugs may be modified by their respective synergists or antagonists.

TECHNIQUE OF BIOMICROSCOPIC EXAMINATION

In the early literature on biomicroscopy there is a paucity of material concerning the conjunctiva. This may have been due to the sudden concentration of attention on the wealth of new data offered by the other optic media. Biomicroscopic examination of the conjunctiva is of paramount value in differentiating between the early signs of trachoma and follicular conjunctivitis. The study of papillary and follicular formations and of early vascular changes in conjunctivitis has been especially revealing. Information may be obtained concerning the layers of the conjunctiva, and the physiologic and pathologic characteristics of conjunctival circulation. Important observations have also been made regarding the tarsal conjunctiva of the everted upper eyelid. However, in order to recognize certain details, it is frequently necessary to use not only special (vital) staining but also to make the observations under the highest magnification (40 \times or more).

METHODS OF ILLUMINATION

I am not in agreement with those who state that the conjunctiva can be examined only by diffuse or semifocal illumination. The

narrow optic section, indirect or proximal illumination, and, to a degree, even specular reflection, may be utilized to advantage.

Diffuse Illumination. By daylight and under low magnification a survey of the entire conjunctiva should be made in order to avoid incorrect judgments concerning the color of the structures. For purposes of comparison, using the same magnification, the conjunctiva should be examined in diffuse illumination obtained with the unfocused beam. The method of Gullstrand has been recommended for this purpose because a larger area of illumination of uniform intensity can be obtained. With the new apparatus of Comberg and Poser,* this type of diffuse illumination is readily afforded by means of the large circular opening in the disk diaphragm.

Direct Focal Illumination. Most observations of the conjunctiva are made in direct focal illumination. With the narrow beam, an optic section of the conjunctiva can be obtained. As the width of the beam is narrowed the intensity of the illumination decreases proportionately. Although this lowering of light intensity is considered a handicap by many ophthalmologists and is overcome by the use of an arc lamp or by overloading the nitra bulb, actually, because it lessens diffusion and reflection, it increases visibility and perception of depth (a process similar to closing the diaphragm of a camera lens or the condensing system of a laboratory microscope). In the hands of Cuénod and Nataf optic section has given excellent results.

Specular Reflection. With this method, zones of intensely brilliant reflexes are seen. The series of conjunctival elevations produce shimmering reflections, comparable to light reflected from ripples on the surface of a body of water. Corpuscular elements, mucus and waxy meibomian secretion in the tear film are observed as these materials are propelled by eyelid action across the field. At times a fine graininess may be discerned on the surface of the conjunctiva, the significance of which is uncertain. With the wide beam, it may be necessary to avoid the zones of specular reflection because of uncomfortable dazzling experienced by the observer.

Proximal or Indirect Illumination. This method of illumination is

* Made by Bausch & Lomb Optical Company.

of small value in examining the conjunctiva. However, when using the narrow beam, adjacent structures, that is, vessels, nerves, scars, will stand out in relief because of the scattered light. Also, because the transparent conjunctiva rests on the opaque sclera, some of the light is reflected back, simulating retro-illumination. Therefore, in examining the conjunctiva, the effects of retro-illumination and proximal illumination may appear intermingled and be indistinguishable.

Diaphanoscopy. Trantas has advocated a technique of transillumination, which is accomplished by everting the upper eyelid over the cone of a Lange transilluminator or a May light.* This examination must be performed in complete darkness. Normally, the silhouette of the meibomian glands and the vascular network may be seen; pathologically, as edema and infiltration increases, the visibility of structures decreases. Consequently, this method is recommended for studying the incipient stages of conjunctivitis. Trantas advises its use in the early stages of trachoma because he believes that it is possible thus to differentiate between papillae and follicles; the papillae appear dark and the follicles more translucent.

Other Methods of Illumination. Bailliart advocated the use of a special lamp which he designed and called "lampe à lumière froide" (cold light lamp). Panico and other ophthalmologists have found a yellow filter advantageous for the illumination of the conjunctiva. Many biomicroscopists have recommended the use of red free light for the examination of the conjunctival vessels. Because filters are absorptive, the conjoined use of an arc lamp in order to obtain greater intensity of illumination may be desirable. Red free light makes the conjunctival vessels appear black on a yellowish green background; hemorrhages stand out dark, in bold relief.

BIOMICROSCOPY OF THE NORMAL CONJUNCTIVA

EXAMINATION OF THE PALPEBRAL (TARSAL) CONJUNCTIVA

The conjunctiva of the upper eyelid is more easily examined with the biomicroscope than is that of the lower eyelid. This is due to

* Obtained by removing the head and caps from the ordinary May ophthalmoscope.

its firmer attachment to the upper tarsus. In the tarsal conjunctiva the following may be distinguished* (Fig. 117; Plate I, figs. 3, 4):

Epithelial Layer. The individual cellular outline of the epithelium

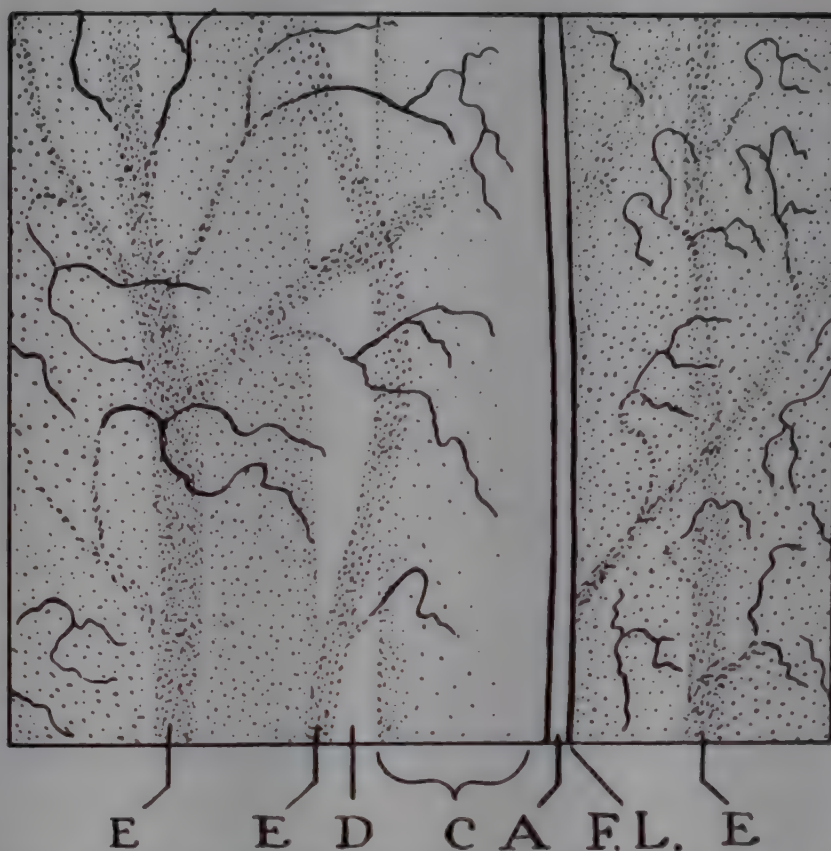


FIG. 117. Optic section through normal tarsal conjunctiva. Diagram of Plate I, fig. 4, F.L., Film line; A, epithelium; C, adenoid layer; D, tarsus; E, deep vessels.

cannot be distinguished unless the conjunctiva is vitally stained (page 149). In spite of the interesting results obtained by many workers, deep vital staining of the conjunctiva is not recommended as a practical routine procedure. On the other hand, the use of weak solutions of fluorescein or Bengal rose to stain the tear film and surface of the conjunctiva is practical and of great aid in localization. With optic section, a drop of 2 per cent fluorescein causes the film to stand out as a sharply stained green line over any portion of the conjunctiva. This type of staining causes the patient no discomfort and is washed away quickly by the eyelid movements and tear fluids. In optic section the epithelial thickness is represented by a thin dark space under the tear film line. The film line is best seen when the lower eyelid is slightly everted; a pool of tears forms in the lower cul-de-sac. As the section passes over this pool, a film line is formed at the point of contact.† This film line continues over the

* The actual layers of the conjunctiva can be seen only with the narrow beam.

† A similar effect can be obtained at the point of contact between the cornea and the upper lid.

conjunctiva and then passes over the cornea as the precorneal film line. In specular reflection a fine graining of the surface of the epithelium, not due to secretion, is frequently observed. In the more diffused light of the wider beam only the subadjacent layers can be seen, owing to the thinness and transparency of the epithelium.

Subepithelial Layer. The color of the subepithelial tissue of the tarsal conjunctiva as seen in optic section varies somewhat, depending on the length of time the eyelid is everted. For example, immediately on eversion of the eyelid, the color changes from white or gray to yellow; after prolonged eversion, it changes to a yellowish red owing to congestion. Under high power the subepithelial tissue seems to be composed of a granular material suspended in a grayish matrix of varying density. The deepest portions are more opaquely yellow, merging indistinctly with the intransparent subconjunctival tarsal tissue. The fibrous layer is absent in the tarsal conjunctiva.

The Vessels. The larger vessels are easily seen even with the unaided eye and, depending on their size, appear orange, pink, or red in color against a grayish pink background. In optic section they are localized in the subepithelial layer. From eight to ten small vessels, called "short vessels," derived from the perforating twigs of the internal arcade (marginal or juxtaciliary arcade of Terson), emanate from the upper margin of the everted eyelid. Other vessels, equal in number but larger and longer, may be seen on the edge of the everted eyelid at the free tarsal boundary (lower margin) (Plate I, fig. 1). When this margin is normal it resembles a straight clean-cut line, whereas irregularities are observed in the early stages of all types of conjunctivitis. Behind this margin the long vessels, from the peripheral arcade of Terson, appear. The long and short vessels anastomose freely in the inferior third of the tarsus, forming the third zone, called the zone of anastomatic network. This region always seems paler than its surrounding areas. The final ramifications of the network anastomose and form a plexus of remarkable regularity. The fine meshes of this capillary plexus form a polygonal or hexagonal design.

In a perfectly normal conjunctiva there are neither papillae nor follicles. As the examination is prolonged, the observer will see brilliant glistening reddish points, which are vascular buds (incipient papillae) produced by the slight irritation from eversion of the eyelid.

Follicles are often observed in young children of lymphatic habitus; these follicles are always situated at the lateral ends (external or internal) of the tarsus, and their presence may point to adenoidal pathologic changes.

The conjunctiva of the lower eyelid, although presenting a smaller surface, is similar in structure to that of the upper eyelid.

EXAMINATION OF CONJUNCTIVA OF THE FORNIX

Biomicroscopic examination of the fornices, especially the upper fornix, is arduous, because of the difficulty in obtaining a good exposure of the parts by eyelid eversion. Moreover, owing to the thickness of the conjunctiva and the folds resulting from its loose attachment to the underlying structures, it is not easy to obtain a good optic section.

EXAMINATION OF BULBAR CONJUNCTIVA

For the purposes of description the bulbar conjunctiva can be divided into two zones: (1) the scleral zone, and (2) the perilimbal zone. Both zones are easily examined with the biomicroscope.

The scleral portion of the bulbar conjunctiva is somewhat loosely attached, being separated from the episcleral tissue by a potential space, which permits gliding movements of the tissue over the bulbar surface except near the limbus where the conjunctiva is more firmly attached.

As with the tarsal conjunctiva, it is possible to obtain an optic section of the bulbar conjunctiva (Fig. 118). The film line thus rendered visible is best delineated when stained with fluorescein (Plate II, fig. 1). Below the film line there is a narrow, dark, non-relucent and nonrespersive linear space, representing the epithelial thickness. The subepithelial layer (adenoid layer) is indicated by a diffuse grayish white area, delicately stippled, reminding one

of a cloudy colloidal suspension. In adults, this layer may become more densely granular owing to the presence of whitish linear particles of varying sizes, while in the aged it becomes compressed,

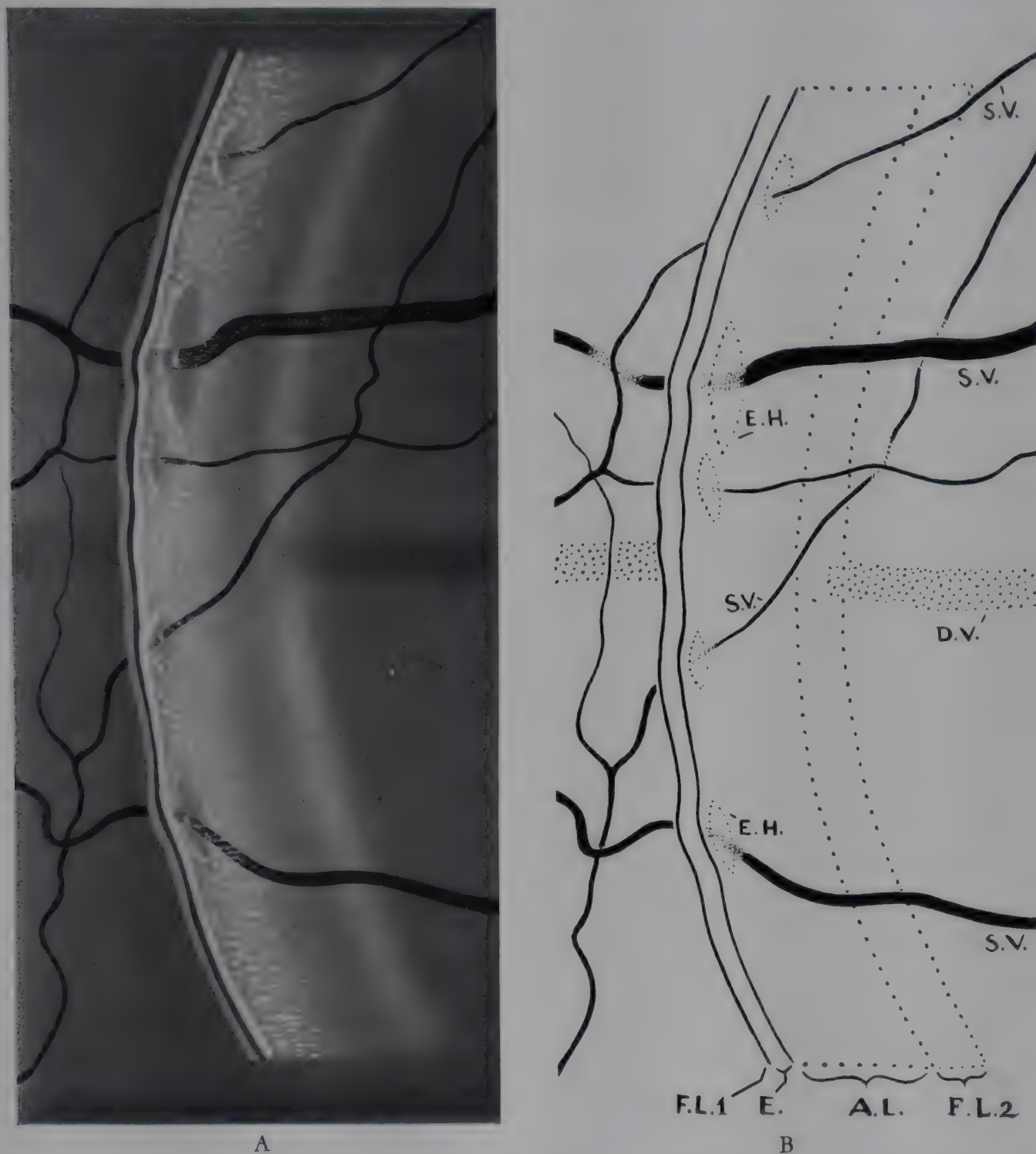


FIG. 118. A. Optic section through the normal bulbar conjunctiva. B. Schematic drawing of A showing layers in bulbar conjunctiva, *F.L.*, Film line; *E.*, epithelium; *A.L.*, adenoid layer; *F.L.2*, fibrous or hyaloid layer; *S.V.*, superficial vessels; *D.V.*, deep vessels; *E.H.*, elliptical halo seen around vessel.

narrow, and less relucant* (Plate II, fig. 2). Beneath this area is the denser, opaque, and yellowish fibrous layer which merges indistinctly with the nontransparent episclera.

* The degree of opalescence of the adenoid layer governs in part the degree of conjunctival transparency. However, some form of turbidometry would have to be employed in order to make any worth-while deductions concerning relative degrees of opalescence.

PLATE II

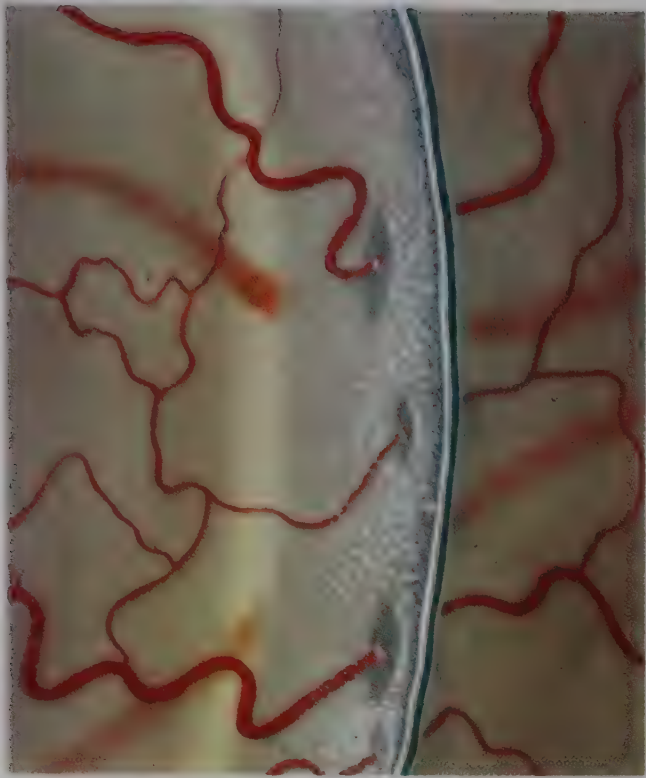
FIG. 1. Optic section through the bulbar conjunctiva in a 17-year-old girl. 40 X.

FIG. 2. Optic section through bulbar conjunctiva in individual, aged 55, showing thinning of the conjunctiva; increased density of the deep part of the adenoid layer which obscures the underlying episcleral vessels. 40 X.

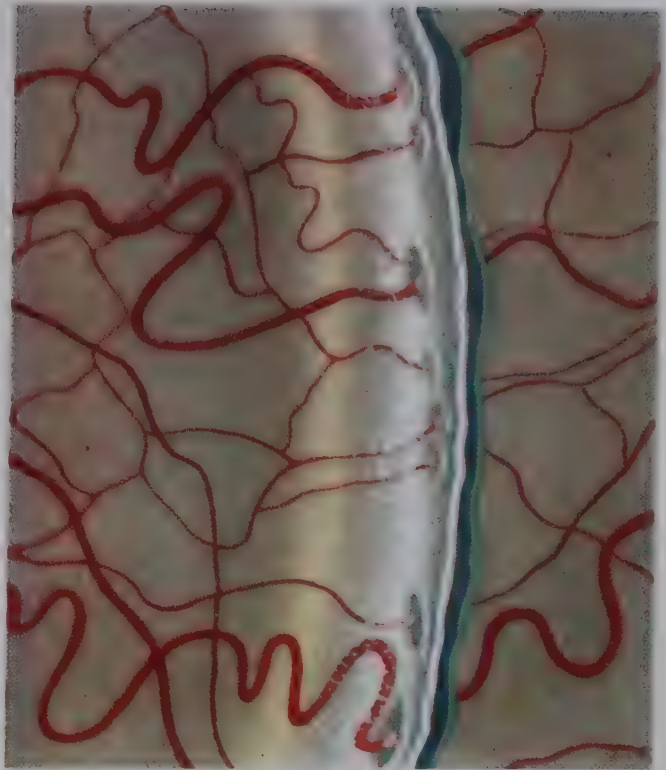
FIG. 3. Normal limbus. Diagrammatic photograph of Figure 4. *I*, In optic section; *II*, by retro-illumination; A, limbal spur; B, palisade; C, palisade (retro-illumination); D, terminal arcades and zone of physiologic edema.

FIG. 4. Normal limbus.

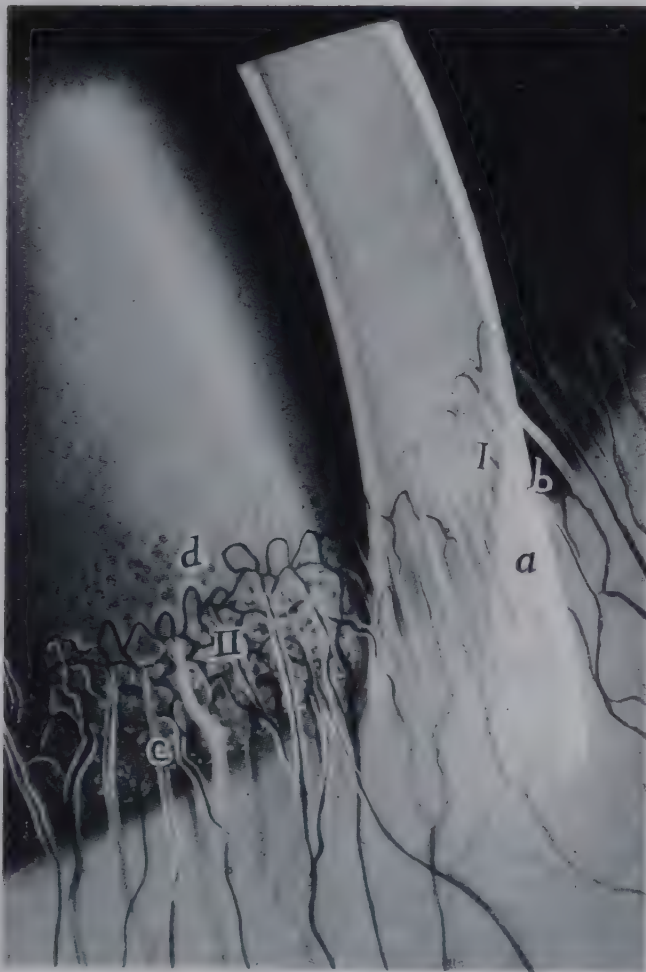
FIG. 5. Conjunctival limbal pigmentation in the aged.



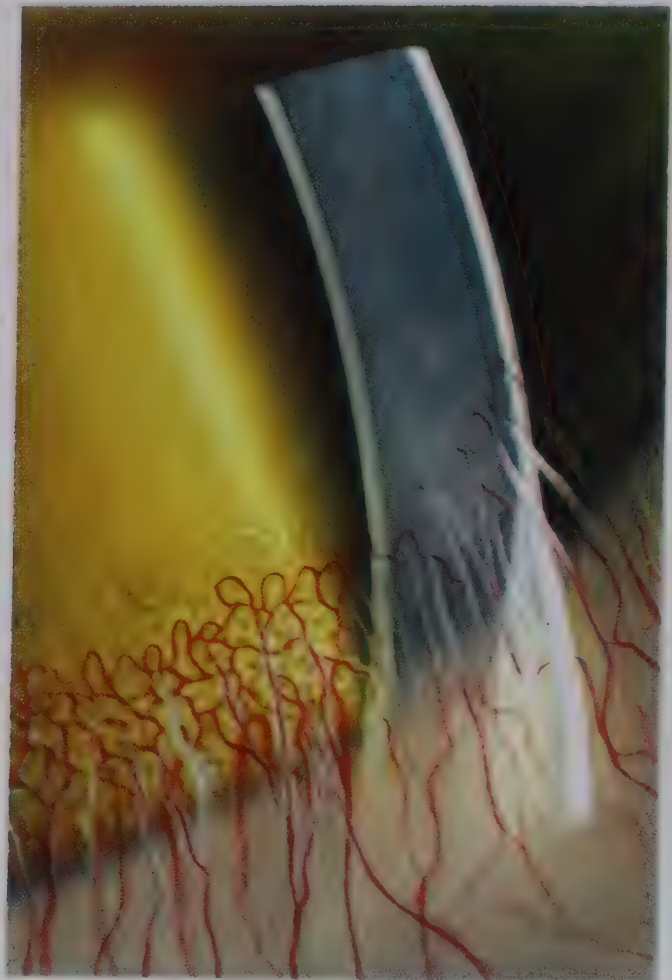
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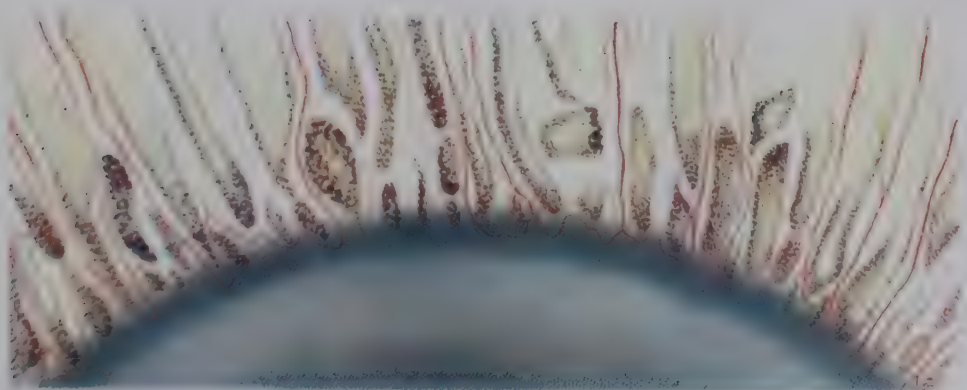
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The biomicroscope readily reveals the two layers of conjunctival vessels: (1) the superficial, situated in the subepithelial layers, and (2) the deep, situated in the fibrous layer adjacent to the episclera. The superficial vessels, arteries as well as veins, are a brilliant red color; the deep vessels, larger in size, have a pink or a violet hue. Although anastomosis may occur, the two layers are approximately 0.5 mm. apart. The superficial vascular network is composed of numerous small anastomosing vessels branching in all directions.* When one of these vessels lies just below the epithelium, it raises the latter slightly causing an abrupt elevation of the film line. Frequently, a dark elliptically shaped area encircles vessels in the adenoid layer, probably representing a zone of lesser density. The clearness with which the deeper vessels are seen depends on the transparency of the flocculent adenoid layer and varies from case to case and with the age of the individual. Large episcleral vessels may be visible when this layer is transparent or thinned by age or when these vessels are congested. They appear as pinkish bands and tend to lie in a single plane. At times, a vessel may emerge from the sclera almost at right angles to its surface. At the point of perforation a small grayish collar of translucent tissue is seen encircling the emerging vessel. In most cases, each arterial vessel is accompanied by only one vein. The circulation can be discerned in some of the smaller venous branches as quick waves, or as a fragmented column. Other vessels reveal only an immobile blood column.

EXAMINATION OF THE LIMBAL REGION

The biomicroscopic appearance of the normal limbus varies from case to case, according to the age of the individual, vascular architecture, presence of palisades, and pigmentation. In addition, its appearance varies according to the method of illumination employed (Plate II, figs. 3, 4).

In direct focal illumination (optic section) one can see the superficial limbal spur quite well (*q. v.*); in sclerotic scatter it can be

* In most persons over forty these vessels become more prominent. Likewise, in drawing conclusions concerning them, one must also take into account the frequency of mild conjunctival irritations, resulting from eyestrain, exposure to common physical agents (sun, dust, wind, smoke, and the like).

made out as a faint haze; while in retro-illumination or proximal illumination, it is hardly discernible. In order to obtain good retro-illumination of this zone it is best to have the beam directed from the opposite side (e.g., when examining the temporal limbus, one should direct the beam from the nasal side). In optic section, the spur may be seen clearly as a gradually diminishing wedge of relucant tissue, the apex of which fuses with Bowman's zone and the base of which merges imperceptibly with the sclera (Plate XVII, fig. 2). By retro-illumination or proximal illumination this wedge-shaped region appears relatively transparent and gives the impression of an abrupt demarcation between the cornea and sclera, which does not actually exist. The terminal vascular arcades lie immediately in front of the superficial scleral spur.

In many individuals, direct focal illumination or retro-illumination reveals a series of short brilliant white radiating lines, known as palisades (Plate I, fig. 5). It should be emphasized that these structures are not seen in every patient. With retro-illumination or proximal illumination, the palisades appear in young persons as double-contoured tubes of a gelatinoid nature; while in the old, they become whiter and less translucent. The site of these structures is subepithelial. In some of the aged there is a certain degree of interanastomosis (simulating a network), in the interstices of which pigment is deposited. According to Vogt, the palisade zone varies from 1.0 mm. to less in width, and the distance between palisades is about 0.15 mm.

In optic section, with high magnification ($40\times$) a small afferent vessel may be observed to lie either over or within a palisade. However, in many instances, no vessel can be seen. We have observed in quiescent cases of interstitial keratitis, as well as in injected limbus from other causes, that when palisades are present each palisade contains a vessel. One gets the impression that the palisades at their corneal ends fuse with the apex of the limbal spur where it joins Bowman's zone, and that their conjunctival ends are continuous with the subepithelial connective tissue. It might be conjectured that these structures are hyalin extensions of Bowman's membrane to the basal membrane of the conjunctival epithelium.

Between the white striae there are relatively dark spaces, which show, when vitally stained, a lining of small colored points; these spaces are closed toward the cornea and open peripherally. Some

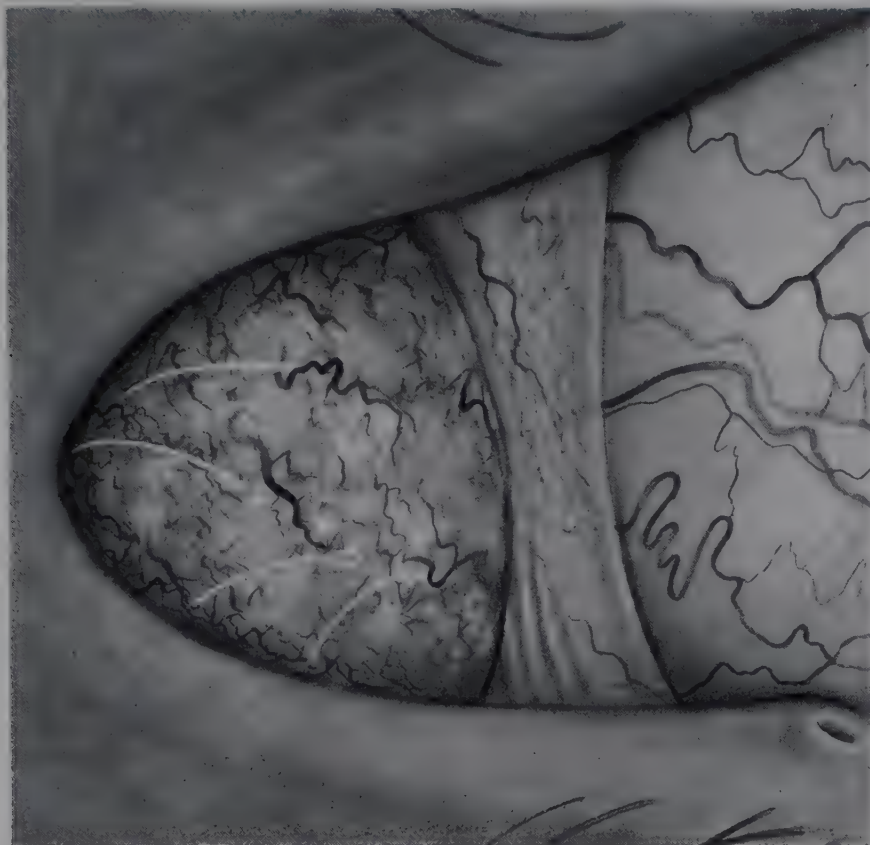


FIG. 119. Lacrimal caruncle and plica semilunaris.

writers have ascribed a lymphatic origin to these structures. Pigmentation along the palisades is likely to occur in the aged, and in dark Caucasians and Negroes (Plates II, fig. 5; IV, figs. 1, 2, 3). In cases in which palisades are not present the circumcorneal conjunctival pigmentation seems to follow the course of the blood vessels.

Furthermore, the existence of a zone of epithelial bedewing in the cornea about the terminal loops of the vascular arcade can be found in every normal eye. These fine droplets are smaller than those characteristic of epithelial edema in pathologic conditions (Plate II, figs 3, 4).

EXAMINATION OF THE LACRIMAL CARUNCLE AND PLICA SEMILUNARIS

The lacrimal caruncle and plica semilunaris may be of particular biomicroscopic interest in conjunctivitis, in new growths, in post-

operative cicatricial alterations, and especially in trachoma (Fig. 119).

At the inner canthus the conjunctiva is thrown into a crescentic fold, which has a concave border toward the globe. In the young, this fold is prominent. The conjunctiva is then reflected over the small, red, fleshy caruncle, which lies in the lacus lacrimalis. Certain aspects of the structure of the caruncle are revealed by the biomicroscope. As many as twenty short colorless hairs may be seen protruding medially from its surface. Here, numerous white spots, probably representing the small sebaceous glands, may also be seen. The caruncle is highly vascularized and with high magnification a dense capillary network is visible.

EXAMINATION OF THE LYMPHATICS

The question as to whether or not a well-defined lymphatic system in the conjunctiva can be seen with the biomicroscope has received a great deal of attention and has evoked many conflicting opinions. Various techniques have been employed to enhance the visibility of lymphatics. The instillation of dyes has given poor results. Subconjunctival injection of dyes, especially brilliant cresyl blue, has been recommended by Cuénod and Nataf. Hydrogen peroxide and dionine (lymphagogues) have also been used. The general opinion is that there are two lymphatic systems, one ensheathing the blood vessels and the other independent. Both systems seem to anastomose freely (Plate III, fig. 4).

VITAL STAINING OF THE CONJUNCTIVA

Because of the accessibility of the conjunctiva and the ease with which it is observed, many biomicroscopists have attempted to stain it with vital dyes. Credit must be given Knüsel and Vonwiller for their original work in this field. Among their first followers were Gallemaerts, Kleefeld, Meesmann, and Koeppe. Many of the aniline dyes used in staining ordinary histologic preparations have been employed. Whether or not vital staining is actually accomplished is still a moot question. It may be that only devitalized or semi-

devitalized elements can be stained (Plate III, fig. 1). However, it is known that in some instances, for example, in the case of nerves, it is possible to stain the living cells. Whether a chemical combination results or whether it is merely a physical phenomenon is not certain, but some fixation of the dye occurs. Because of the doubt that still exists about the exact nature of these processes, deductions concerning observations thus made should be guarded.

Vital staining is accomplished by means of repeated instillations of watery solutions of aniline dyes into the conjunctival sac. Three instillations of two drops each at five minute intervals are usually sufficient.* Although anesthesia may be required for certain patients who experience burning sensations, it is usually possible to instill most dyes without anesthetics. It must be stressed that certain anesthetics, particularly cocaine, produce physiologic changes. Pesme and Sierra, basing their opinion on the work of A. Lumiere, criticized Knüsel and Vonwiller on this point; they maintained that the reason that the epithelium took the stain was due to alterations in these cells caused by cocaine.

Some dyes, particularly methylene blue, are irritating and have a prolonged staining action, lasting four or five days.† I agree with the opinion of Cuénod and Nataf that the best results are obtained with a 0.5 per cent aqueous solution of azur ii, an eosin-methylene blue compound. However, certain dyes seem to have a selective affinity for special tissue; the epithelium is best stained by 0.5 per cent azur ii or 1 per cent methylene blue. The stained cells appear as groups of fine colored points arranged in a netlike pattern. Under high magnification, irregular dotlike deposits outline single epithelial cells or groups of epithelial cells (Plate III, fig. 2).

Subepithelial tissue can be stained by cresyl blue or azur ii. The devitalizing action of cocaine is desirable in order to allow the dye to penetrate. Small star-shaped cells are seen in front or behind the superficial blood vessels. A contrasting effect can be obtained by

* For some reason, freshly prepared solutions of dyes do not act as well as "old" solutions. The solutions should be kept in a dark place for two months before being used.

† After using methylene blue, I make it a practice to close the eye with a dressing for twenty-four hours.

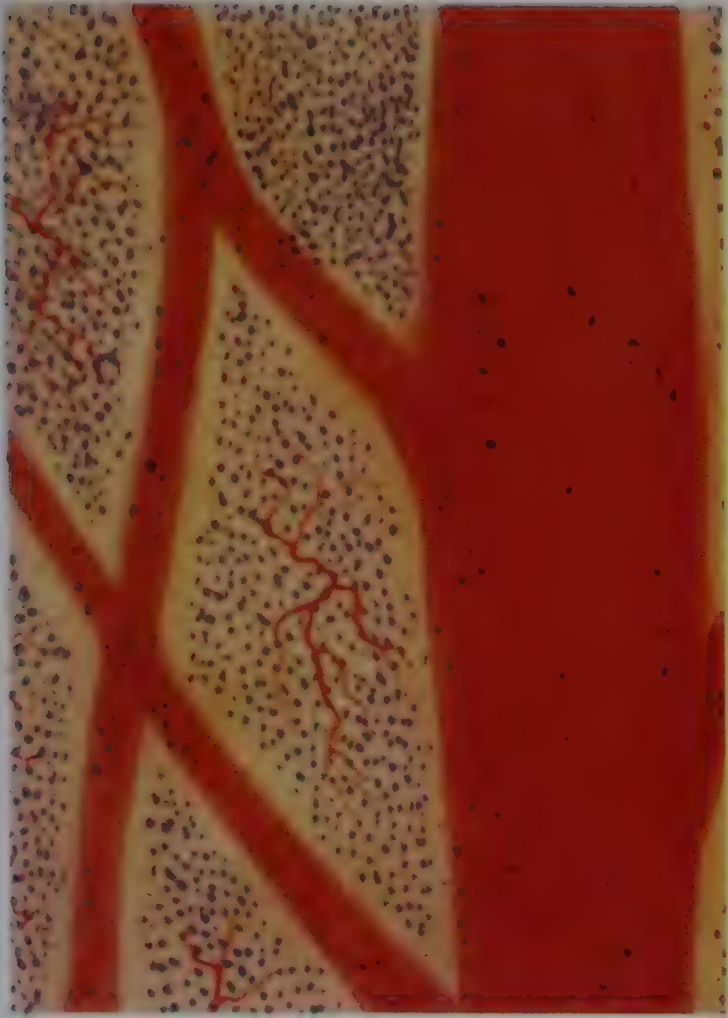
PLATE III

FIG. 1. Normal bulbar conjunctiva stained with azur blue. (After Knüsel and Vonwiller.)

FIG. 2. Vital staining of the conjunctiva showing blood vessels, lymph channels (green), and perivascular sheathes. (After Knüsel and Vonwiller.)

FIG. 3. Vital staining of the conjunctiva with methylene blue showing nerve filaments and endings (60 \times). (After Knüsel and Vonwiller.)

FIG. 4. Vital staining of the conjunctiva showing lymph channels (green). The intermediate punctate dots in Figures 1, 2, and 4 are due to partial staining of the epithelial cells.



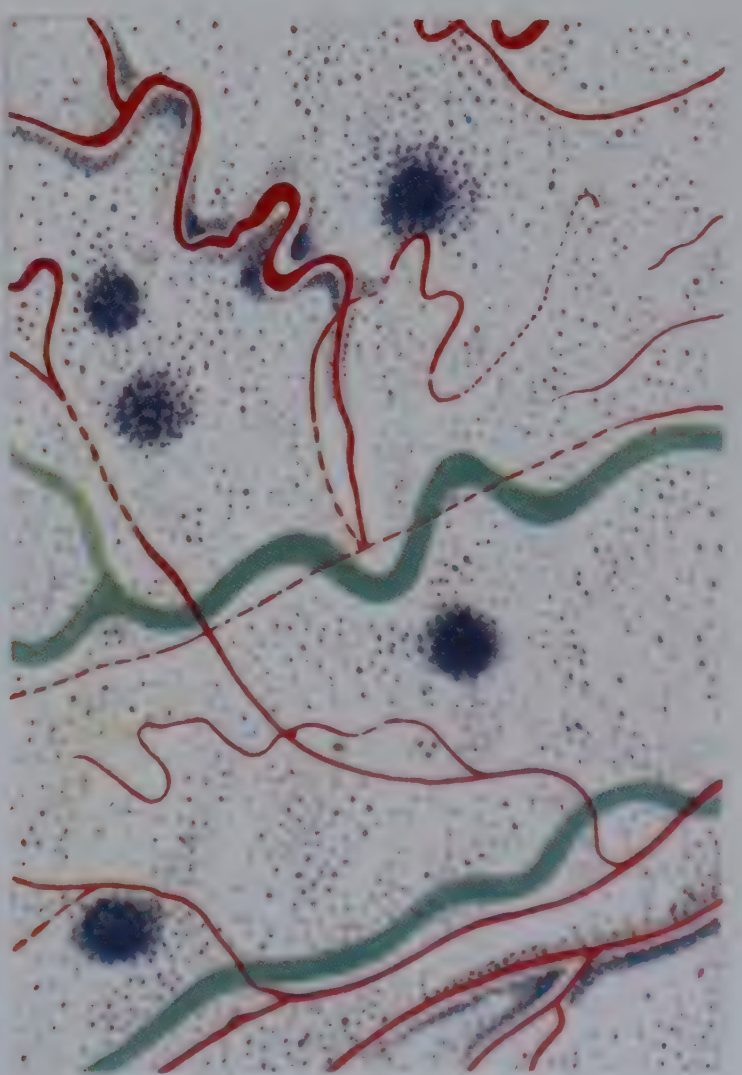
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using 0.5 per cent scarlet red in combination with the dyes just mentioned. This makes the tear film line appear red in contrast to the deeper blue staining. I prefer to use a solution containing 2 per cent of either fluorescein* or rose Bengal for surface staining.

Methylene blue (1 per cent) is the most satisfactory dye for staining nerves; it causes them to appear as delicate bluish threadlike filaments (Plate III, fig. 3). Since nerves take the dye more rapidly than other tissues, they stand out sharply against the colorless background. On the tarsal conjunctiva of the upper eyelid, and in the regions of the upper bulbar conjunctiva and near the limbus, the end plates of the nerve filaments (Krause's corpuscles) can be seen, when so stained. They are globular in shape, measuring from 40 to 100 microns and appear like the ravelled end of a thread. The nerve filaments can also be stained with a polychrome methylene blue dye.

Knüsel and Vonwiller¹⁷¹ state that they have stained the contractile elements surrounding the capillaries, the so-called cells of Rouget. They describe them as being dark and resembling a small "caput medusae."

Occasionally one may be able to demonstrate the presence of a small loop of an *intrascleral nerve*. Loops of this type are seen perforating the sclera and are always covered by freely movable conjunctiva. These loops have been studied by Fuchs and Reese, who described their clinical histologic appearances. Most of them have been found on the nasal side, 3 to 4 mm. from the limbus, although rarely one has been observed inferiorly, superiorly, or temporally. A loop may be flush with the scleral surface or it may be raised somewhat like a mushroom or it may be flexed on itself. As it comes through the sclera it is usually surrounded by a small gelatinous cuff similar to a vessel emissarium. Intrascleral nerve loops are asymptomatic and do not require therapy.

* Although we commonly speak of staining with fluorescein, it should be remembered that this substance does not actually stain tissue but rather acts by the method of diffusion.

Chapter Five

NONINFLAMMATORY LESIONS OF THE CONJUNCTIVA

DEVELOPMENTAL ANOMALIES OF THE CONJUNCTIVA

ISOLATED developmental anomalies of the conjunctiva are rare. In most cases there are associated congenital defects of the entire ocular apparatus. Mann²¹⁰ classified these anomalies thus: (1) absence of conjunctiva — cryptophthalmia; (2) partial metaplasia; (3) pigmentation; (4) anomalies of arrangement — folds, bands, accessory caruncle, hyperplasia of the plica semilunaris; (5) congenital tumors.

PARTIAL METAPLASIA

In partial metaplasia the conjunctiva assumes the character of skin with hair follicles and sebaceous gland formation. This has been observed in occasional cases of dermoids or nevolipoma.

PIGMENTATION

Conjunctival pigmentation is not a common finding in the white races, except at the limbus or caruncle. However, in the colored races a considerable amount of conjunctival pigmentation is usually present and in many instances flecks of pigment may be seen in the bulbar conjunctiva (Plate IV, figs. 1, 2). Occasionally even in the white races such deposits may be found in the bulbar conjunctiva (but these are histologically different from nevi). Although usually considered harmless, cases have been noted in which such flecks suddenly increased in size and spread malignantly over the conjunctiva.

Changes of a locally malignant nature have developed from an innocent-appearing pigment clump at the limbus.

Diffuse pigmentation of the conjunctival epithelium is rare.

Melanosis oculi, occurring in nonpigmented individuals, is characterized by a marked increase of pigment in the uveal tract as well as in the sclera. The conjunctiva is not involved. The color of the sclera may vary from dark gray or purple to deep brown, depending on the location and amount of pigment and the "veiling" effect of the overlying nonpigmented conjunctiva. This point may be demonstrated by the use of the optic section (Plate IV, figs. 3, 4, 5, 6).

Anomalies of conjunctival arrangement are uncommon and differ considerably in each case. They vary from a localized symblepharon to epitarsus (apron-like folds of the conjunctiva attached to the inner tarsal surface of the eyelid). Congenital pterygium and pingueculum have been reported. Congenital hypertrophy of the plica semilunaris with cartilage development may occur as an atavism corresponding to the nictitating membrane in lower animals.

TUMORS

Among the principal congenital tumors occurring on the surface of the eye (epibulbar) are dermoids and dermolipoma.

Dermoid Tumors. Dermoid tumors are most frequently situated at the limbus either in the bulbar conjunctiva or on the cornea, and occasionally in both tissues simultaneously. The most common site of occurrence is the upper, outer quadrant. The caruncle may also be involved.

Although these tumors are usually small at birth, there is considerable tendency to further growth during childhood, and pubescence may be marked by growth of discrete hairs on the surface. Most commonly these tumors are fixed to the underlying tissues; they are opaquely yellowish in color, ovular and flattened horizontally. With the naked eye the surface appears smooth; but in optic section, a granular structure may be located below the epithelium. Hair follicles and tiny glandular concretions may be present.

The sole indication of a dermoid growth may be congenital

PLATE IV

FIG. 1. Normal pericorneal melanotic pigmentation in the Malayan.

FIG. 2. Normal limbal pigmentation in a Negro with beginning arcus senilis (optic section).

FIG. 3. Optic section of conjunctiva in melanosis bulbi showing location of pigmentation in sclera.

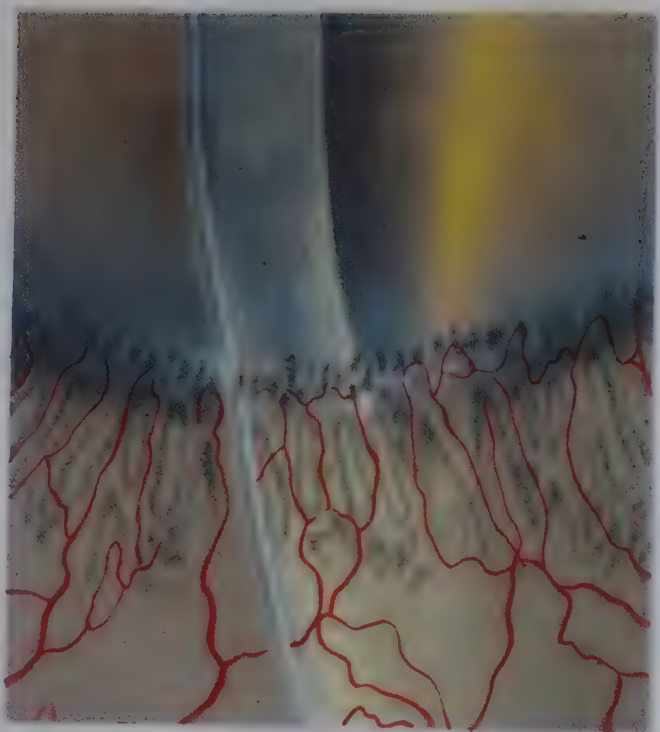
FIG. 4. Melanosis bulbi. Arrangement of the pigment about the deeper vessels. Diffuse illumination. Same case as Figure 3.

FIG. 5. Melanosis bulbi by direct focal illumination.

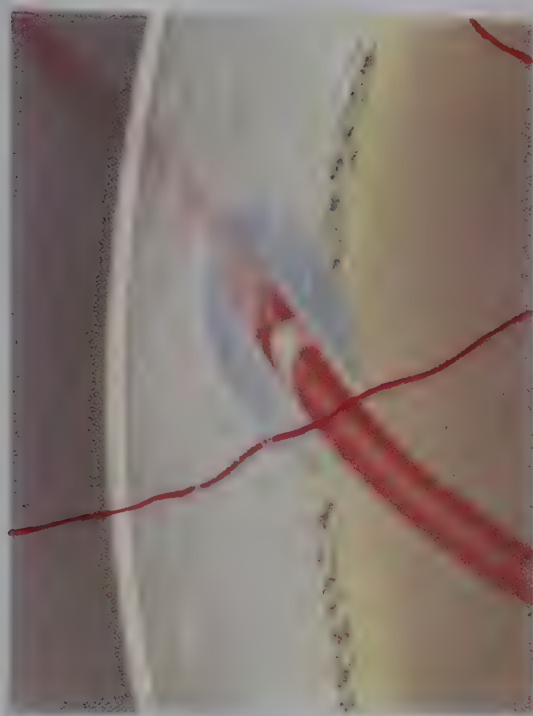
FIG. 6. Diffuse illumination. Distribution of pigment is especially marked about the episcleral vessels. Same case as Figure 5.



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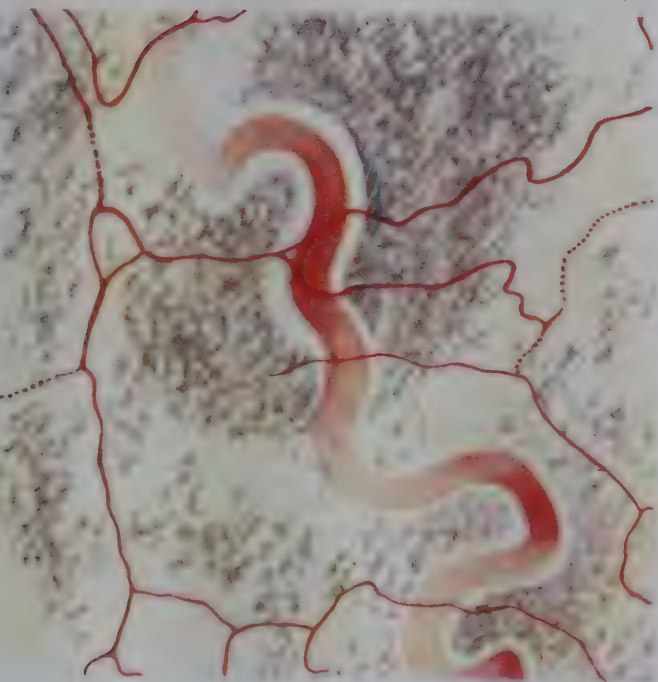


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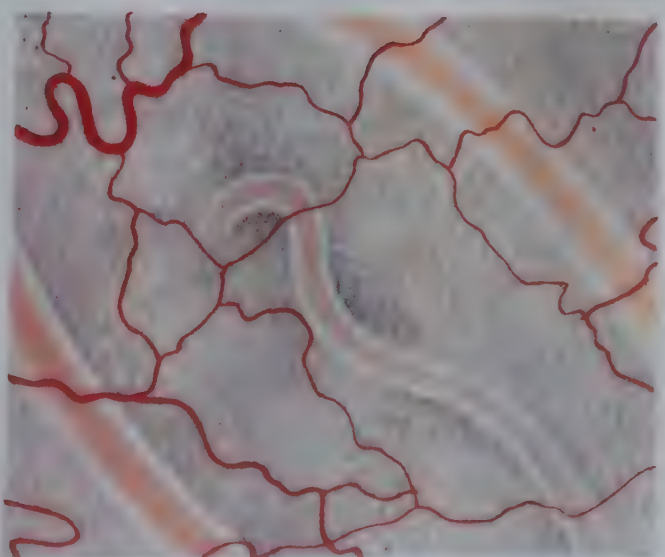
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epithelial plaque formation on the bulbar conjunctiva. This consists of much thickened keratinized epithelium situated near the limbus and appearing as a small, dense, irregular, dirty-white patch which lacks the characteristic conjunctival transparency and luster.

Dermolipoma. A form of dermoid, called dermolipoma because of the high concentration of fatty elements, is yellowish and opaque in appearance and may be mistaken for the rarely seen true lipoma. It is usually subconjunctival, deeply attached, and occurs in the same site as the dermoids. Proof of its dermoid character may require histologic examination.

DEGENERATIVE CHANGES OF THE CONJUNCTIVA

CHANGES ASSOCIATED WITH SENILITY

The conjunctiva undergoes degenerative changes with age, similar to those that take place in other tissues of the body. Whether or not it is correct to consider the physiologic aging processes as true degeneration depends on one's concept of senescence. There is no doubt that heredity plays an important role in the life cycle of every cell, and that degenerative changes similar to those in senility may occasionally appear early in life (abiotrophy). However, metabolic disorders may play a role in these degenerative changes. Because the histologic changes which occur in the aging process often simulate those that result from other noninflammatory degenerations, they are included in the category of the so-called primary degenerations (noninflammatory).

With increasing age the conjunctiva becomes thinner, and as a result of changes in texture and color there are varying degrees of loss of transparency. The bluish white color of the eyeball gradually acquires a yellowish tinge during middle life; this is most noticeable in the deeper layers of the conjunctiva. These changes are particularly prominent in the exposed portions of palpebral fissure, frequently forming a raised horizontal band, from 2 to 3 mm. wide, extending laterally from either limbus. In the older age groups, changes occur in the conjunctiva that are similar to those found in mild cases of prexerosis and consequently may be mistaken for this

condition. In the former there are loss of luster and transparency owing to elastic, hyaline, fatty and cystic changes; raised areas, vaguely similar to Bitot's spots, may be seen as well. The raised spots (pinguecula) do not have the characteristic foamy character of true Bitot's spots, possibly owing to the fact that the overlying epithelium is not affected to the same degree that it is in xerotic states. Whether such "aging" changes are actually similar to those which occur in prexerosis or are due to the same causes which produce prexerosis or even xerosis, is still unknown. However, as a rule the alterations which occur with age are not accompanied by marked subjective symptoms such as photophobia, lacrimation, or feelings of irritation. The larger superficial vessels raise the epithelium, become more prominent, occasionally showing tortuosities and varicose dilatations. Telangiectases and small angiomas, resembling petechial hemorrhages under low magnification, are frequently seen. Also loss of transparency, due in part to the increase of relucency of the thinned, compressed adenoid layer, may obscure the view of the deeper vessels. Attenuation of the limbal capillaries has been described by Vogt.⁸²²

Calcareous deposits are a common finding in both the tarsal and bulbar conjunctiva of the aged (Fig. 120). They appear as tiny yellowish white hard deposits, either single or multiple, sometimes surrounded by a vascular network. In the beginning, they are seen in optic section to lie beneath the epithelium but in many instances, following erosion, the deposits may project above the surface and thereby cause irritation.

Yellowish brown pigmentation surrounding the limbus, especially in the exposed area of the palpebral fissure, may be seen in the thinned subepithelial layer. Vogt considers it hematogenous in origin. The pigment is adjacent to the vessels, but not in direct contact with them, being separated by a clear perivascular space.

Although with age there is, histologically, thickening of the epithelium with a slight tendency toward keratinization, the subepithelial tissue atrophies and undergoes hyaline degeneration with loss of elastic fibers so that the entire thickness of the conjunctiva is actually decreased. With the biomicroscope this change is indicated

by narrowing of the adenoid layer and loss of light transmission to the deeper fibrous layer (Plate II, fig. 2).

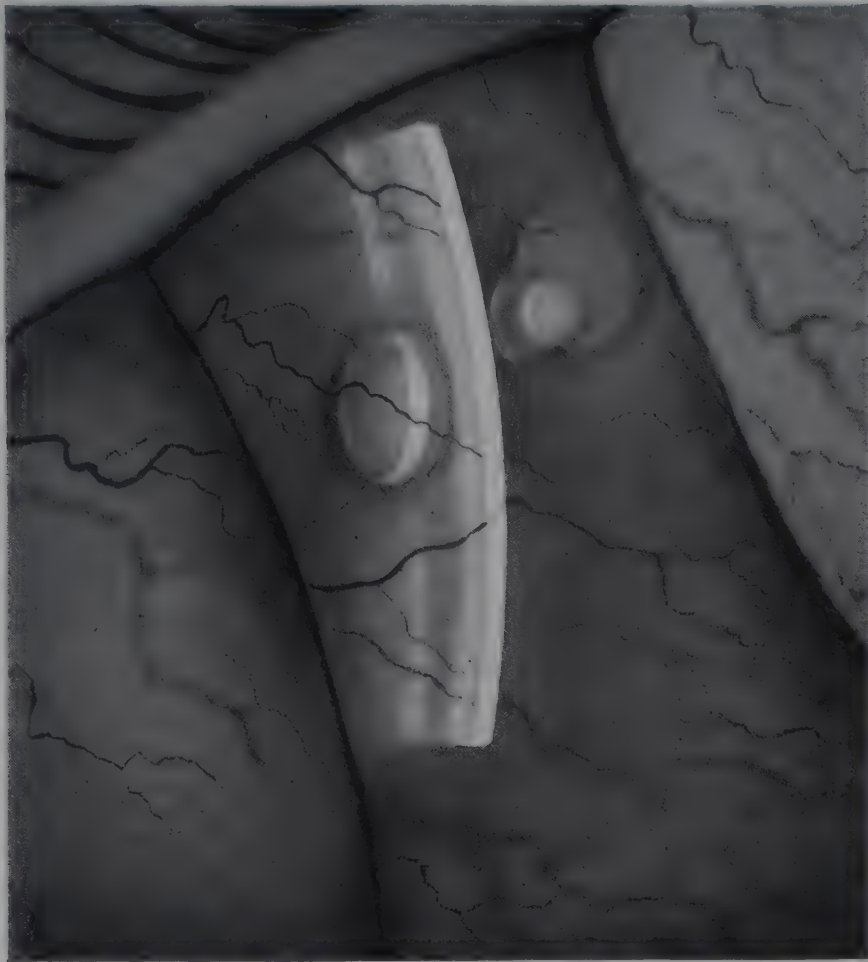


FIG. 120. Conjunctival concretions in the aged near the caruncle.

PINGUECULA *

This is a common degenerative condition which occurs with age (Plates V, fig. 3; VI, fig. 6). It appears as a small white or yellowish, more or less triangular patch situated on the bulbar conjunctiva along the horizontal meridian in the exposed interpalpebral zone on either side of the cornea. When small, such lesions are usually round. Pathologically, the degenerative change is hyaline infiltration of the subepithelial layers and proliferation of elastic fibers. Fuchs maintained that pinguecula is the forerunner and cause of pterygium. Its yellowish tinge is due to the elastic tissue and not to fat as originally believed. The biomicroscopic appearance of a pinguecula is as follows: By direct focal illumination, an irregular raised grayish granular or gelatinous subepithelial patch is seen within the depths

* The symmetrical appearance of pinguecula in uniovular twins as described by Vogt would indicate that this presenile or senile condition is genetically determined.

PLATE V

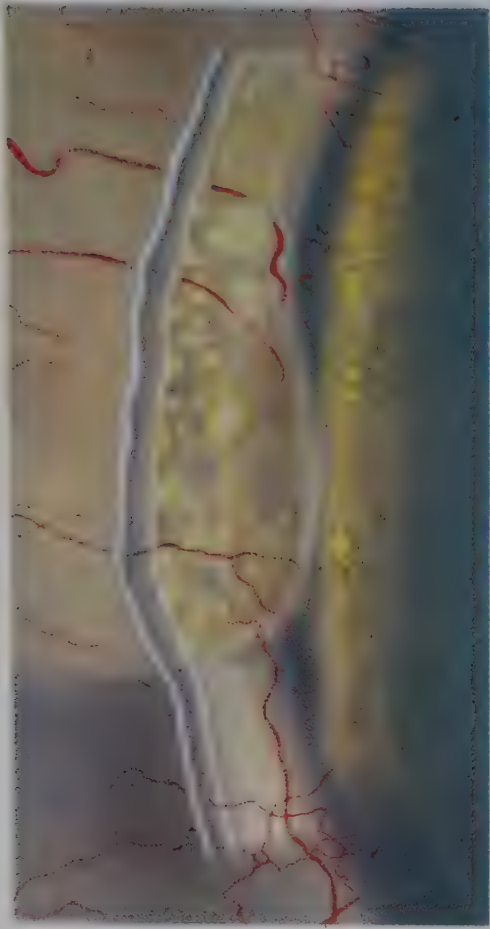
FIG. 1. Xanthomatous patch on the bulbar conjunctiva in a case of Gaucher's disease. (After B. Friedman.) Direct focal illumination. Optic section shows the deep situation of the lipin deposits in the conjunctiva and arc-like formation of deposits in the corneal periphery.

FIG. 2. Xanthomatous patches (same case as in Figure 1) illuminated by fluorescent light. (After B. Friedman.)

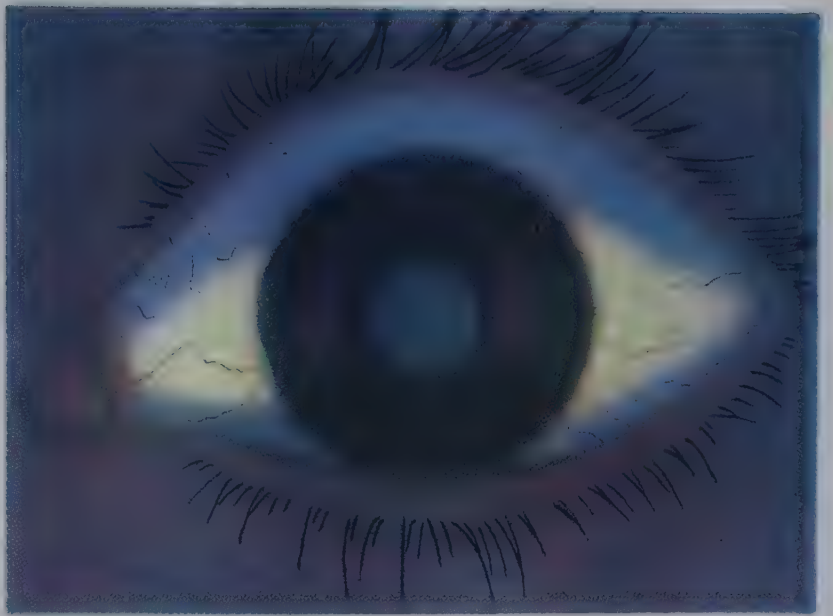
FIG. 3. Pingueculum in optic section stained with azur ii and counterstained with fluorescein, illustrating hyalinization in the deeper layers.

FIG. 4. Bitot's spot.

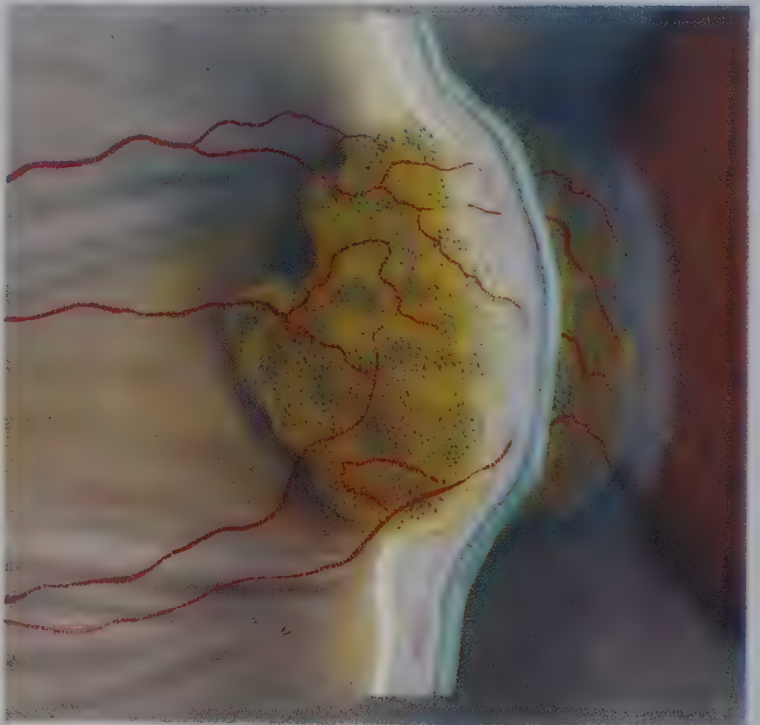
FIG. 5. Optic section through a subconjunctival hemorrhage, showing onset of clearing stripes.



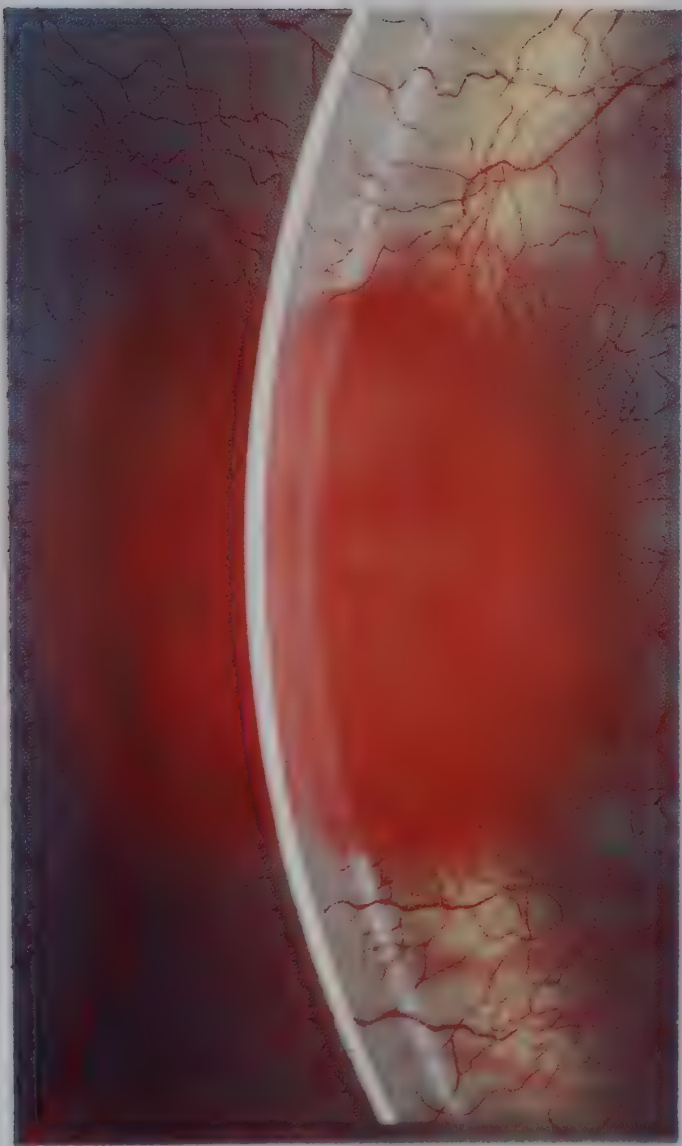
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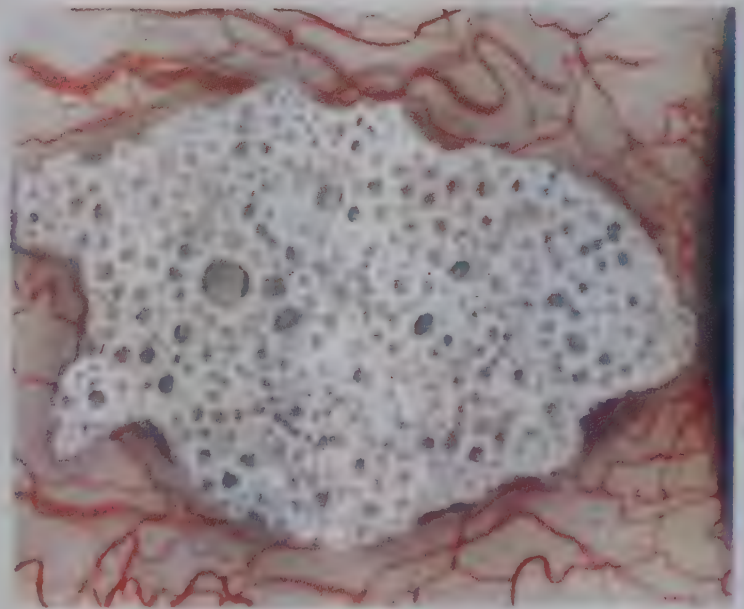
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of the optic section; at its border irregular yellow spots impart a yellow tinge. There may be small cystlike cavities over the surface and within the maze itself. In optic section, staining with azur ii and counterstaining with fluorescein produces a beautiful picture of its structure (Plate V, fig. 3).

FATTY AND LIPOID DEGENERATION

Small yellow concretions may frequently be observed on the bulbar conjunctiva (interpalpebral zone) of elderly persons; they usually result from degenerative cellular products, occurring in depressions or within folds (pseudo-glands) of the conjunctiva. Fatty infiltration, according to von Szily,³³¹ produces an orange-yellow tinge in the conjunctiva. Elschmig and Stanka described nodules in the conjunctiva due to fat infiltration, following the prolonged use of ointments. Such nodule-containing fat globules on the tarsal conjunctiva may be seen with the biomicroscope. They usually cause a low-grade inflammatory reaction. *Xanthomatous patches* in the conjunctiva have been discerned in certain conditions, such as Gaucher's disease and Christian-Schüller disease; these are associated with increased storage of lipid.

Some cases of Gaucher's disease present a characteristically hypertrophied pinguecula. The growth usually appears on the medial side and later develops on the temporal side. It is colored a deeper yellow than the ordinary pinguecula and also shares in the brownish discoloration which is evident in the skin of these patients. The mass is several times the size of the pinguecula seen in the normal eye, and may even overhang the limbus slightly. Friedman,⁹⁹ reporting on the biomicroscopic appearance of such lesions, states: "Examination discloses numerous yellowish globular masses, some discreet, others coalescent. Many of the globules are pinpoint in size, but others assume a diameter of a millimeter or more. The tissue between the globules is granular, and contains a large number of grayish and dark brown particles. True fat is absent in the pinguecula of the normal eye and also of Gaucher's disease. Since the latter contains the specific foam cells associated with the disease,⁷⁶ it is fair to

assume that it also contains the cerebroside kerasin which is found in the other affected organs. The yellowish infiltration extends a short distance into the adjacent cornea, just below the level of Bowman's membrane. Flattish yellow droplets are visible; these coalesce in spots to form sheet-like deposits (Plate V, fig. 1). Numerous dark brown granules are interspersed between the infiltrations. The blood vessels in the case pictured were wider and more irregular in their diameter than normal. The ordinary pinguecula emits a chartreuse color in fluorescent light; in Gaucher's disease this fluorescence is startlingly accentuated" (Plate V, fig. 2).

XEROSIS OF THE CONJUNCTIVA

Xerosis of the conjunctiva may be a sequel to a local disease or to exposure or it may be a symptom of malnutrition. It is no longer considered to be caused by drying due to lack of tears, but rather to result from actual changes in the tissue. In the early stages there is a loss of luster, as demonstrated by specular reflection, while later, owing to loss of elasticity, a wrinkling of the conjunctiva occurs with the formation of folds (Plate XI, fig. 1). These folds are curved concentrically to the cornea; in their furrows may be found tears and secretion. Near the limbus, small, definitely outlined dry patches covered with a peculiar foamlike substance, so-called Bitot's spots,* may be seen (Plate V, fig. 4). Bitot's spots, which occur principally in children, generally are triangular in shape, the base directed toward the limbus and the apex toward the canthi. Tobgy²⁹⁸ has described them as sharply outlined, white in color and having surfaces covered by a foamlike substance which can easily be removed but which quickly reforms after a few hours. The lacrimal secretion does not adhere to the spots; these appear dry and raised above the surface of the conjunctiva. When small, they are usually oval or circular in shape; as they increase in size they assume the characteristic triangular shape in the interpalpebral fissure. They are

* Bitot's spots must not be confused with pinguecula. In the former the surface epithelium is definitely affected, whereas the latter are always situated deeply in the conjunctiva and are covered by normal epithelium (Berliner²⁶).

covered by a thickened or modified epithelium. According to the literature (Palmer), Bitot's spots may appear in a variety of forms. Nicholls and Nimalasuriya have suggested that there are two forms of xerosis in the conjunctiva: (1) an acute type with the formation of foamy spots and (2) a type in which white striations occur. Metivier²²⁴ states: "Sie-Boen-Lian (1938) observed 19 cases of Bitot's spots in Java in which deficiency of vitamin A was clinically and biologically excluded; and he came to the conclusion that Bitot's spots are by no means a positive sign of deficiency of vitamin A. He says that the majority of his patients affirmed that they had the spots in early childhood and even believed that they were present from birth. Similar statements have been made to me by Trinidad parents and young adults. This difficulty or impossibility of curing Bitot's spots with vitamin A preparations in certain groups of patients is fairly well recognized among persons with pigmented skins, for Palmer, Sie-Boen-Lian, Aykroyd, and Rowland Wilson (personal communications, 1938), Nicholls, and I have had experiences more or less the same, in Assam, Java, South India, Egypt, Ceylon, and Trinidad. The records of observers in different parts of the world have been collated and brought into relationship with experience gained in Trinidad in order to bring out the full meaning of Bitot's spots and other related conjunctival appearances." In mild cases (*prexerosis conjunctivae*), there may be only a moderate loss of luster and transparency with wrinkling and Bitot's spots possibly absent; but when severe, the conjunctiva acquires dermoid characteristics.

Pillat²⁴¹ observed with the biomicroscope small pigmented dots and clumps in the optic section of the conjunctiva resulting from avitaminosis A. He localized brownish dots of pigment (probably melanin) in the epithelial layers and larger clumps of pigment below these dots in the subepithelial layers. This pigmentation, which seems to be a late manifestation, is attributed to a prolonged deficiency of vitamin A and is apparently peculiar to the Orient. Corneal involvement and night blindness are also associated with this condition (Chapter V). It is still questionable whether individuals

having mild or moderate deficiencies of avitaminosis A will exhibit definite conjunctival changes, such as loss of luster, transparency, or Bitot's spots. In a small series of cases in infants, I was unable to find any conjunctival changes; these patients were known to be deficient in vitamin A by blood tests.

ENDOGENOUS PIGMENTATION OF THE CONJUNCTIVA

BLOOD PIGMENTATION

Following the absorptive stage of a subconjunctival hemorrhage, groups of delicate yellowish brown crystals of hemosiderin may be detected in or below the epithelium.* Associated with these crystals, there may be small, almost black, amorphous granules which are derived from the hematoidin moiety. Severe hemolyzing diseases, such as malaria, yellow fever, and acute septicemia, frequently cause yellowish staining of the conjunctiva owing to the liberation and deposition of blood pigment derivatives. Many severe febrile disorders may be accompanied by conjunctival hemorrhages (Plates V, fig. 5; VI, figs. 1, 2).

BILE PIGMENTATION

In icterus, the conjunctiva is stained by the bile pigments carried by the blood stream. In icterus of the newborn the marked yellow staining may even extend to the conjunctival secretion.

MELANIN PIGMENTATION

Pigmentation of the conjunctiva as an anomaly has been described (page 154). In pathologic states there may also be deposition of melanin. The deeper cells of the conjunctival epithelium are potentially melanoblastic and consequently the exposed portions of the conjunctiva, particularly near the limbus, are likely to become pigmented under certain conditions. This may happen in association with systemic conditions, in which there is a generalized pigmentary disturbance (e.g., Addison's disease), and also in local degenerative

* These can be seen only with the biomicroscope.

diseases of the conjunctiva, such as xerosis conjunctivae, trachoma, and vernal catarrh. Pigmented deposits are also seen in nevi, neoplasms, and cystic degeneration of the conjunctiva.

Melanin pigment, appearing as brownish or black granules, should be differentiated from the yellowish lipochrome pigments of the bilirubin type which are found in the lipoidoses of the Gaucher and Schüller-Christian syndromes, as yellowish wedge-shaped thickenings (page 165).

ADDISON'S DISEASE

In Addison's disease the presence of fine granular pigment deposits in the epithelial and subepithelial tissues has been reported by Meesmann²¹⁹ and by others. These deposits tend to form a dark concentric circle in the exposed limbal regions. Löewenstein¹⁹⁶ reported a case in which long continued instillation of epinephrine produced pigmented nodules in the conjunctiva.

OCHRONOSIS *

Sallman²⁶⁰ and Cuénod and Nataf⁵¹ described a scleroconjunctival perivascular pigmentation occurring in ochronosis, a disease which is characterized by brown or black discoloration in the cartilages, tendons, and skin, and by arthritis and alcaptonuria. The pigmentation results from a disturbance of metabolism of the amino acids, namely, tyrosin and phenylalanin, precursors of melanin. A case I saw recently through the courtesy of Dr. J. W. Smith exhibited a bulbar pigmentation of the subconjunctival and episcleral layers, notably at the tendinous insertions of the lateral recti muscles (Plate VI, fig. 3). Deep in the conjunctiva, the pigment, brown in color, was deposited within tiny cystic lymphatic dilatations in a circular manner, forming rings and hemispheric figures, while over the tendinous

* In three cases seen by Dr. J. W. Smith, the pigmentation involved the cartilages of the auricles, giving them a cyanotic appearance. The ocular pigmentation seems to advance slowly, depending on the progress of the disease which tends to run a long and protracted course. In the youngest patient, aged 8, the pigmented layer was barely visible. In the oldest, it was easily discernible with the unaided eye as elliptically shaped black deposits, 2 mm. in width and 5 mm. in length, situated about 4 mm. from the corneal limbus, and running somewhat concentric to it. The neighboring conjunctival vessels were slightly engorged but not disproportionately so for individuals of this age.

PLATE VI

FIG. 1. Subconjunctival hemorrhage near the limbus with clearing stripes around vessels. Direct focal illumination.

FIG. 2. Same case as Figure 1. Diffuse illumination and higher power. Residual perivascular pigmentation is seen near lower right hand corner of illustration.

FIG. 3. Optic section of conjunctiva in ochronosis. Away from the heavily pigmented areas the pigment granules are arranged in signet-ring formations.

FIG. 4. Argyrosis of the conjunctival fornix. Pigmentation occurs in the sub-epithelial layers.

FIG. 5. Vascular congestion of the bulbar conjunctiva near the limbus in a case of acute conjunctivitis.

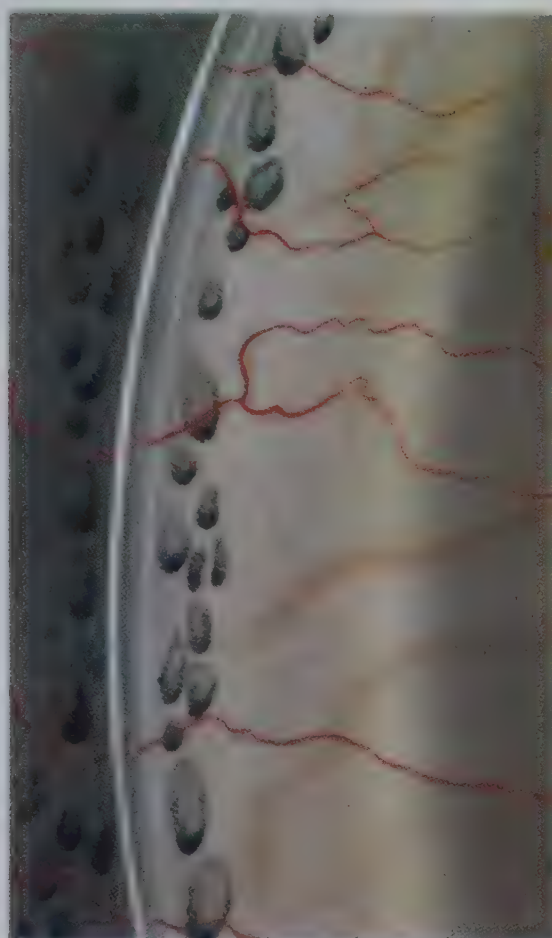
FIG. 6. Pingueculum by diffuse illumination.



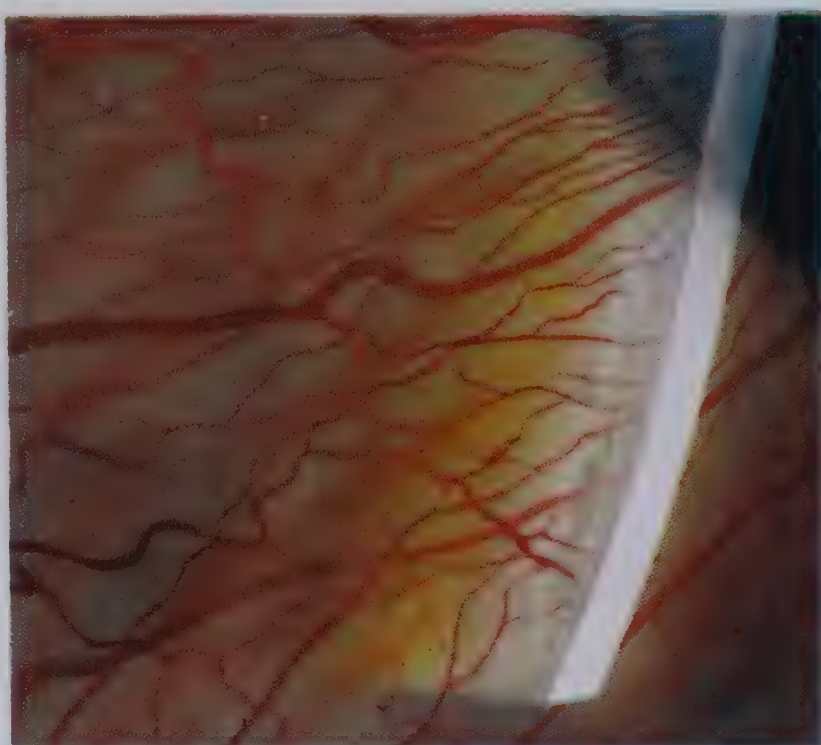
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insertion of the recti, it was deposited in dense slate colored elliptically shaped masses in the episcleral and scleral tissues (Fig. 121 B). In addition, at the corneal periphery many small isolated

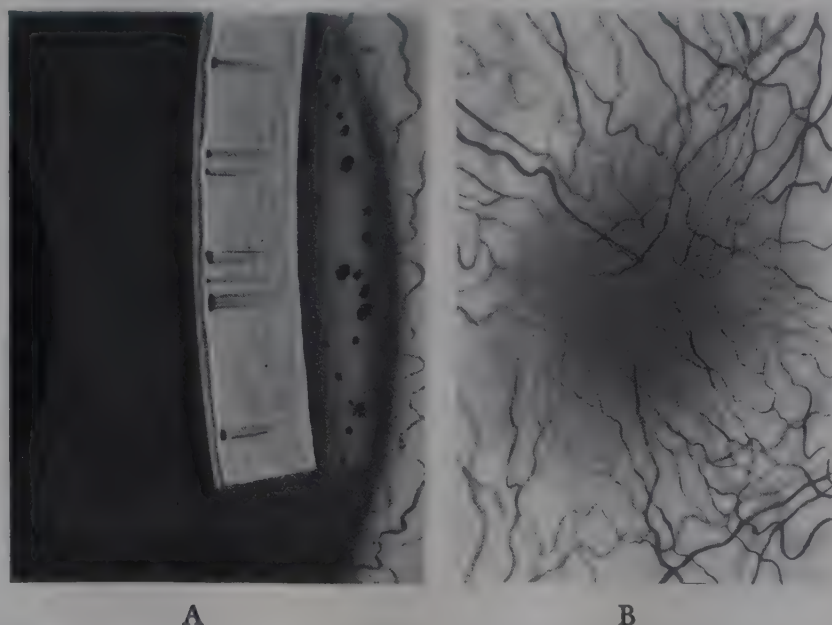


FIG. 121. A. Ochronosis. Pigment deposits resembling oil droplets at limbus and in cornea (Bowman's zone). B. Deep conjunctival and scleral pigmentation in ochronosis.

pigment clumps resembling oil globules were seen below the epithelium (Fig. 121 A) (Bowman's zone) in the exposed portion of the palpebral fissure.

It is interesting to note that such substances as adrenalin, dichlorobenzene, dinitrophenol, homogentisic acid, and tyrosine are derivatives of benzene. When these are ingested, they tend to form phenol derivatives in their intermediate metabolism. These intermediate products, at the slightly alkaline pH of the body fluids, tend to polymerize into pigments. This can be seen in adrenalin when it is left exposed to the air. These pigments deposit in such relatively bloodless tissues as the sclera, lens, cartilages, and produce the pathologic deposition of pigment seen in the ochronosis.

VITILIGO

In vitiligo (leukoderma) hyperpigmentation of the conjunctival epithelium has been reported by Hanssen.¹⁴⁴ Histologically, the pigment was found in the basal cells of the epithelium, associated with a deeper-lying inflammatory reaction.

EXOGENOUS PIGMENTATION OF THE CONJUNCTIVA SILVER DEPOSITS

Argyrosis is one of the most common types of conjunctival pigmentation. Formerly, when the salts of silver were employed as an internal medicament, conjunctival pigmentation frequently resulted as a part of generalized argyrosis. Today, it is usually due to the instillation of silver preparations into the conjunctival sac (silver nitrate and colloidal silver compounds, such as protargol, argyrol, and neosilvol). It may also appear as an occupational disease; then, according to Vogt,³²⁵ the pigmentation of the conjunctiva and Descemet's membrane may be its only outward manifestation. In either general or local argyrosis, the silver granules are deposited around the elastic fibers below the conjunctival epithelium, which itself is never invaded. As a rule the silver causes no inflammatory tissue reaction.

In argyrosis which follows instillation of a colloidal silver preparation, Descemet's membrane may exhibit a mottled bluish tinge of a definite pattern caused by the deposition of silver. Friedman⁹⁸ reported a series of cases of conjunctival argyrosis and emphasized the frequency with which Descemet's membrane is impregnated (Plate XXV, figs. 1, 2). Recently, I observed this condition in a young woman who had received instillations of argyrol in the conjunctival sac for a period of two years. The conjunctiva itself was not involved. The diagnosis was made biomicroscopically on the basis of the characteristic bluish tinge of Descemet's membrane.* When impregnated, the conjunctiva has a dull gray, slate color; this discoloration may be seen over the entire bulbar conjunctiva but the precipitates † are most numerous in the regions of the lower fornix, caruncle, and superior tarsal conjunctiva. This may be due to the fact that since these areas are most exposed, they receive the first full impact of the medication. With the biomicroscope, the stain-

* Undoubtedly corneal impregnation is frequently overlooked, because clinically it is difficult to discern it except by biomicroscopic examination.

† Chemically, the granular deposit is probably a reduction compound of silver albuminate. It is never found within the cells, but is adherent to the connective tissue and elastic fibers.

ing is seen to be composed of fine granules situated below the epithelium, following the course of the vessels.* If conjunctival folds of the fornix are affected, the crests of the folds appear dark, whereas the troughs are unpigmented (Plate VI, fig. 4). Koeppe¹⁷⁸ described the formation in such cases of small epithelial vacuoles in the lower fornix, which after rupturing, simulate the appearance of shagreen. The boundaries of the semilunar fold and caruncle when heavily impregnated form a dark limiting line. When the semilunar fold is dusted with a brownish pigment, the caruncle appears dark and the lobules of the caruncular glands are clearly outlined.

SIDEROSIS

Siderosis of the conjunctiva commonly occurs after the prolonged retention of an iron foreign body. The oxidized iron particles are deposited in the neighboring tissues and appear as granular rust-colored deposits. According to Koeppe¹⁷⁷ they have the same distribution as the silver deposits in argyrosis. In one case that I have seen, the pigment seemed to lie in the deepest layers of the conjunctiva where small brown dots were so disseminated as to suggest that the lymphatic network was outlined by the scavenging action of the lymphatic system. Würdemann has stated that a yellowish pigmentation caused by siderosis may follow the long-continued internal use of sulphate of iron.

* Recently Loewenstein¹⁹⁸ demonstrated the subepithelial disposition of the silver particles histologically. He found the epithelium entirely unaffected. Also by instilling a drop of 2 per cent dionine into the conjunctival sac of a patient having conjunctival argyrosis he produced edema and with the biomicroscope discerned a minute grayish brown network situated in the edematous fluid between the sclera and the conjunctival epithelium. He stated: "No lymph vessels were visible either accompanying the blood vessels, or separate; and there was no dust (silver) surrounding its blood vessels suggesting the existence of lymph vessels." This is contrary to the findings of Koeppe.

Chapter Six

INFLAMMATORY AND TRAUMATIC LESIONS OF THE CONJUNCTIVA

CONJUNCTIVITIS

ALTHOUGH many types of conjunctivitis have been differentiated on an etiologic, clinical, or pathologic basis, certain phenomena representing the nonspecific reaction of the conjunctiva to irritation and inflammation are common to all the types.

The discussion of conjunctivitis will be limited to those types in which the presence of special or unusual biomicroscopic features aid in diagnosis. Clinically, it is important to differentiate between conjunctival and ciliary injections. Conjunctival injection is most intense in the region of the fornices and tarsal conjunctiva, gradually decreasing as the limbus is approached; whereas ciliary injection, resulting from a deeper seated disease of the eye, is most intense in the circumcorneal region.

The stages of conjunctivitis may be arbitrarily classified as follows: (1) hyperemia, (2) neovascularization, (3) papillary hypertrophy, (4) follicle formation, and (5) regressive stages.

Hyperemia is the initial change in acute conjunctivitis. It is characterized by injection of the superficial vessels and edema of the conjunctiva (Plate VI, fig. 5). The injection is most intense at the fornix and tarsus, gradually fading toward the limbus; the vessels of the bulb and fornix appear bright red and are increased in size. The tarsal conjunctiva gradually loses its transparency, obscuring the outlines of the meibomian glands. Intensification of the edema may raise the conjunctiva (chemosis), except over the tarsus where the attachment is firm. When the bulbar or fornical conjunctiva

becomes chemotic, separation takes place between the adenoid and fibrous layers. Evidently, it is not possible for fluid to dissect the fibrous layer from the episclera. Imbibition of fluid by the adenoid

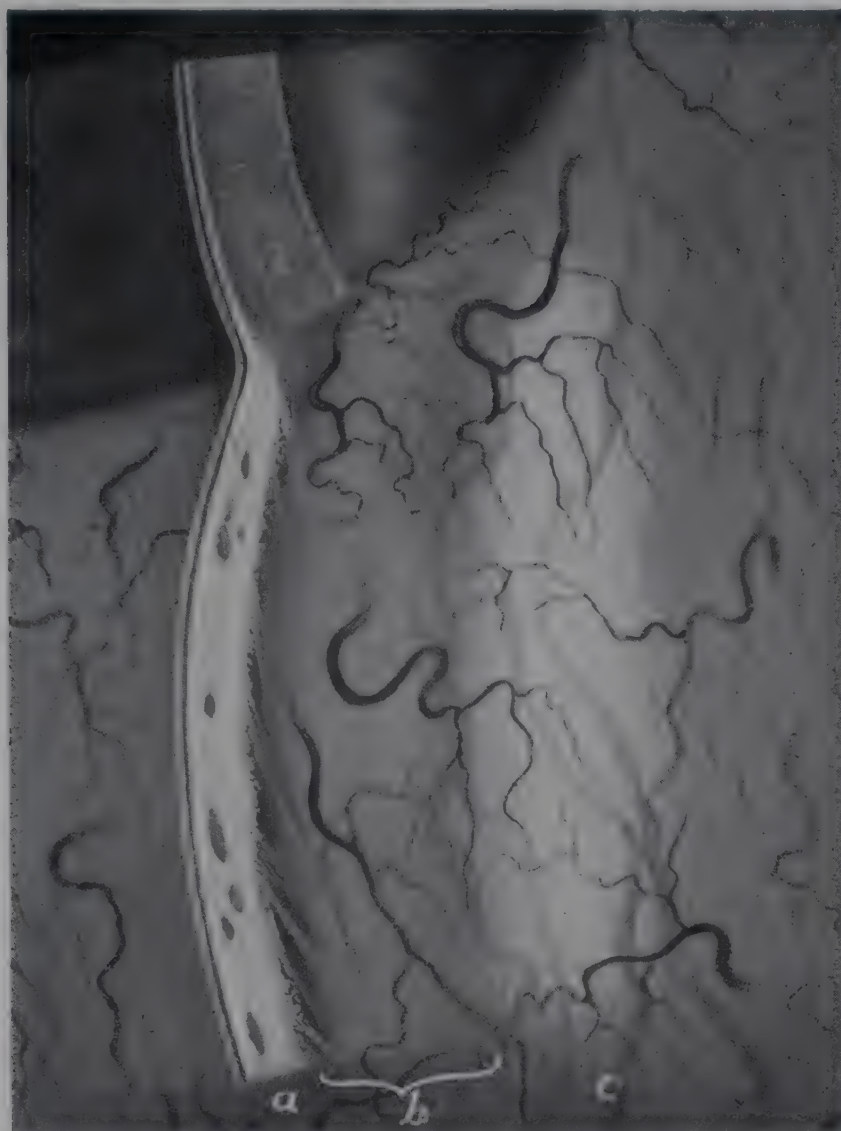


FIG. 122. Chemosis of bulbar conjunctiva. Note the separation of adenoid layer containing water clefts from the fibrous layer. *a*, Adenoid layer; *b*, fluid space separating these layers; *c*, fibrous layer.

layer is represented by the formation of dark clefts or water slits (Fig. 122). The edematous, injected, and raised conjunctiva has a pinkish yellow color. Hyperemia makes it impossible to distinguish the three zones of the normal vascular architecture of the tarsal conjunctiva.

Neovascularization (new vascularization) closely follows the stage of hyperemia and is best observed in the tarsal conjunctiva. From the normal vascular network, situated immediately beneath the epithelium, capillaries proliferate, assuming a course perpendicular

to the plane of these vessels. They proliferate rapidly and open like bouquets or glomus-like tufts in the epithelial layer. With the naked eye, these capillaries appear like little red dots, and may easily be mistaken for petechial hemorrhages. Later, the terminal ends of these microcapillaries, no longer supported by connective tissue, may burst, causing small hemorrhages. Examination of these vascular tufts, by a narrow beam, with intense illumination and high magnification, shows with certainty the origin, the direction, and the starlike disposition of the newly formed vessels. This new vascularization appears only after prolonged mechanical irritation or at the beginning of inflammation (Plate VII, fig. 1).

Papillary hypertrophy. In the first stage of papillary formation, which takes place after neovascularization, the papillae may not be visible, even biomicroscopically, until they cause surface excrescences. With vital staining the outline of these developing structures is indicated by the presence of fine punctate dots, reddish in color, well delineated, and separated one from the other by white lines. When elevated, these dots representing the organization of the newly formed capillary tufts are called papillae and are a constant finding in nearly all types of conjunctival inflammation. Each papilla is due to the organization of one capillary tuft or glomus. (Plate VII, fig. 2). This organization occurs within the normal pre-existing meshes of the adenoidal layer, the histologic structure of which is composed of a polygonal or rounded meshwork from which many bundles of elastic fibers issue to fuse with the basal membrane of the epithelium.

Careful examination with the narrow beam will disclose the fact that each papilla is skeletonized by a tuft of new-formed capillaries, which rise perpendicularly from the plane of the deeper parent vessels, making their way to the surface (Fig. 123). The papillae have a diameter between $1/20$ and $1/10$ mm.; in chronic cases they increase in size, especially toward the free margin of the tarsus; they are separated by threadlike spaces which appear colorless in direct illumination and lightly shadowed in oblique illumination. Cuénod and Nataf believe that these threadlike spaces are the

lymphatic meshwork which follows the course of the smallest blood capillaries.

Vital staining with azur ii indicates the potential outlines of the

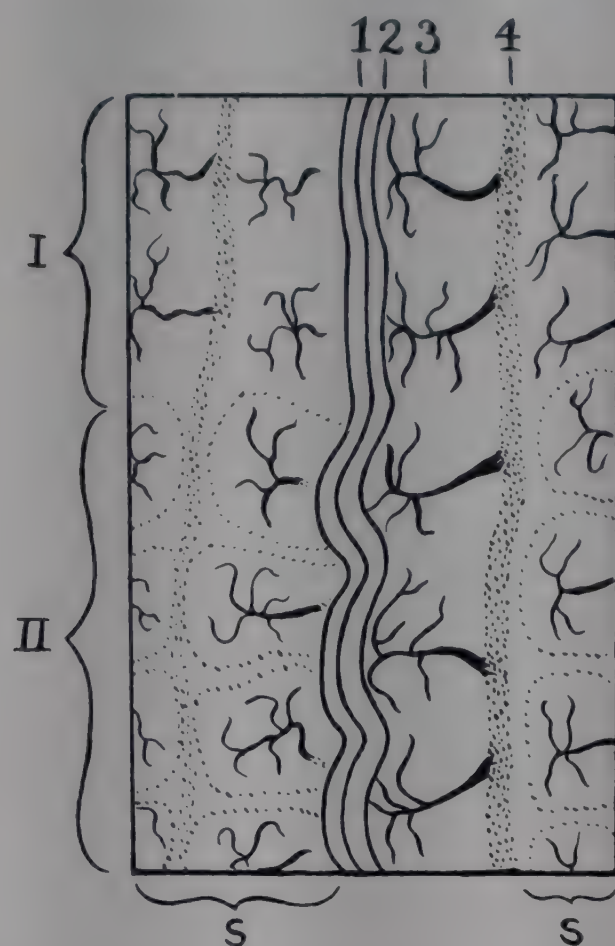


FIG. 123. Diagram illustrating structure of papillae. 1, Superficial epithelial layer; 2, deep epithelial layer; 3, adenoid layer showing vascular buds, developing from large vessels (4) in deeper adenoid or fibrous layer. I, Prepapillary area; II, area of formed papillae. S, Areas outside section (After Cuénod and Nataf.)

papillae even before they are raised above the surface (Plate VII, fig. 3).

It is interesting to speculate as to why the tendency to the formation of papillae occurs in the tarsal conjunctiva and not in the bulbar. Whether this is due to the firm attachment of the conjunctiva to the tarsus, or to some obscure chemotaxis, is still unknown. It may be that there is a directional potential inherent in newly formed tarsal blood vessels, which causes them to take a course perpendicular to the plane of the normal vascular network. At any rate, the progress of newly formed vessels or the filling of previously invisible capillaries in the bulbar conjunctiva is unrestricted since they extend in the same plane as their parent vessels.

PLATE VII

FIG. 1. Tarsal conjunctiva of the everted upper eyelid in acute conjunctivitis (low power). The neovascularization (buds) in early stages of papillae formation.

FIG. 2. Optic section through stained tarsal conjunctiva semidiagrammatic, illustrating vascular bouquets skeletonizing papillae. (Modified after Cuénod and Nataf.)

FIG. 3. Papillary hypertrophy of the tarsal conjunctiva stained with azur ii; the small dark granules result from partial epithelial staining. Diffuse illumination. 60 X.

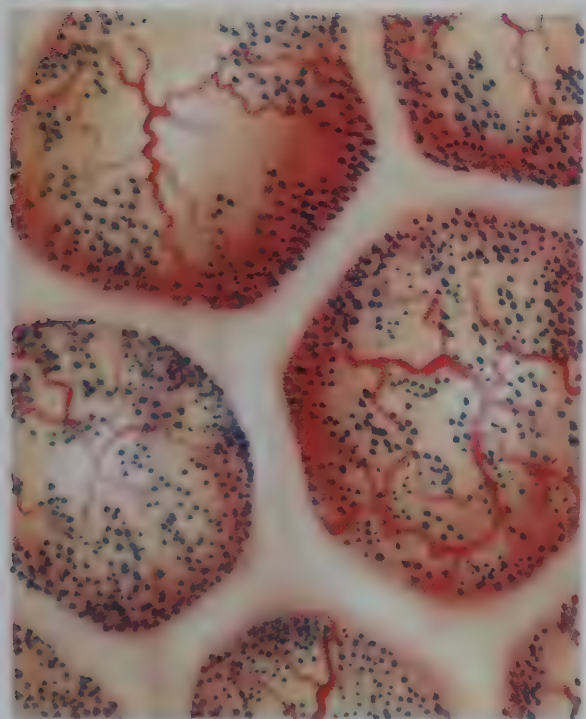
FIG. 4. Acute conjunctivitis (everted eyelid) illustrating congestion, vascular buds and below large horizontal layer of mucoid secretion.



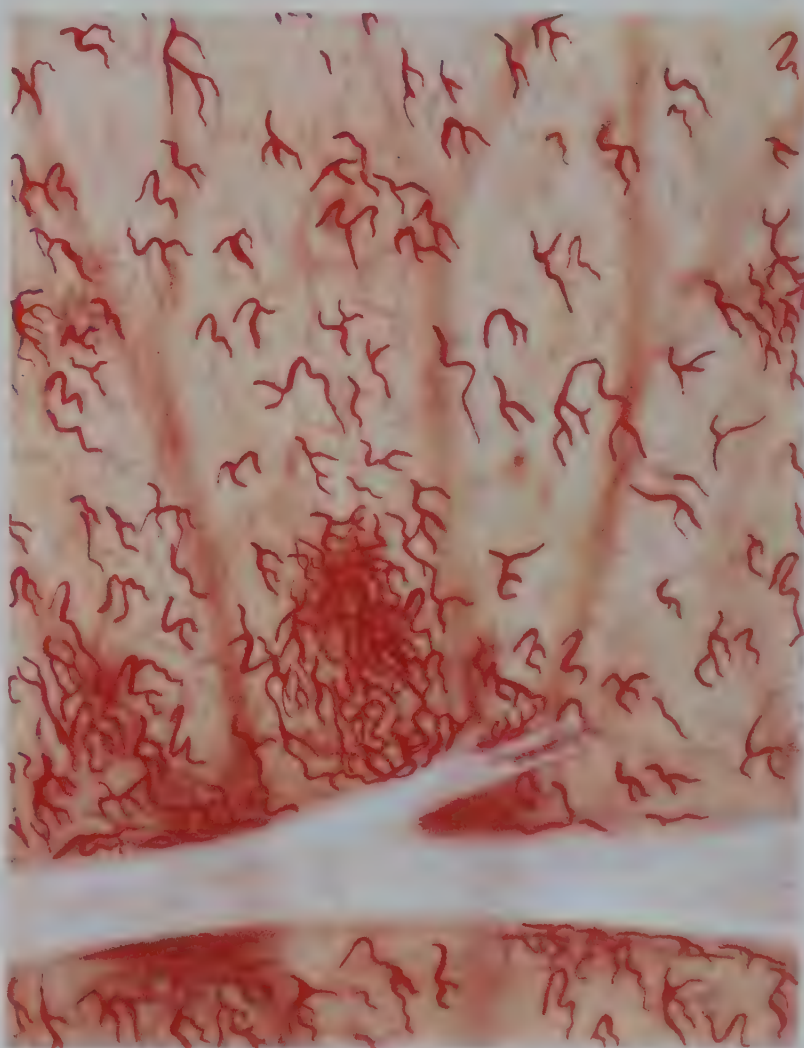
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2



3



4

Follicle formation. As a result of irritation, lymphoid hyperplasia of the adenoidal layer of the tarsal conjunctiva results in the formation of follicles. This reaction is nonspecific and occurs in many conjunctivitides whether of viral, bacterial, or chemical etiology. It must be differentiated from papillary hypertrophy which is the direct result of vascular irritation. Papillary hypertrophy and follicle formation may occur simultaneously. The rate of follicular development is directly proportional to the degree of irritation. However, in children of lymphatic constitution, local conjunctival lymphatic hypertrophy may accompany similar changes elsewhere in the body.

The initial reaction consists of a more or less uniform increase in the lymphoid elements of the adenoid layer, which can be demonstrated only histologically. Examination shows these follicles or nodes to consist of a dense central collection of large mononuclear lymphocytes surrounded by a ring of small deeply staining lymphocytes in the subepithelial tissue. An occasional large vacuolated histiocyte may be found. In later stages the cells aggregate to form a lymph node (follicle). It is at this point in their development that they become visible by causing an elevation of the epithelium (Fig. 124). With the biomicroscope, indication of these changes is manifested by the presence of clear raised areas, which are usually present between the papillae. They are several times larger than papillae and when fully formed, appear as translucent, hemispheric protuberances. The follicle may become quite superficial and the overlying epithelium markedly thinned out. Spontaneous evacuation seldom occurs. In contrast to the central vascular tuft in the papillae, the follicle has a superficial vascular trellis-like network. Small vascular branches are seen to climb over the elevated surface, and at times, before terminating, may penetrate into the depths of the follicle. We see papillary hypertrophy best developed in the allergic types of conjunctivitis while folliculosis is most marked in the viral infections.

Regressive stages. Although in their early stages there is no difference in the appearance of the follicles of initial trachoma, of the follicular conjunctivitides, or of those resulting from sensitivity to

atropine or physostigmine, the ultimate fate of follicles may differ. Ordinarily, follicles as well as papillae diminish in size as time goes on and eventually disappear, leaving no trace. However, in trachoma,

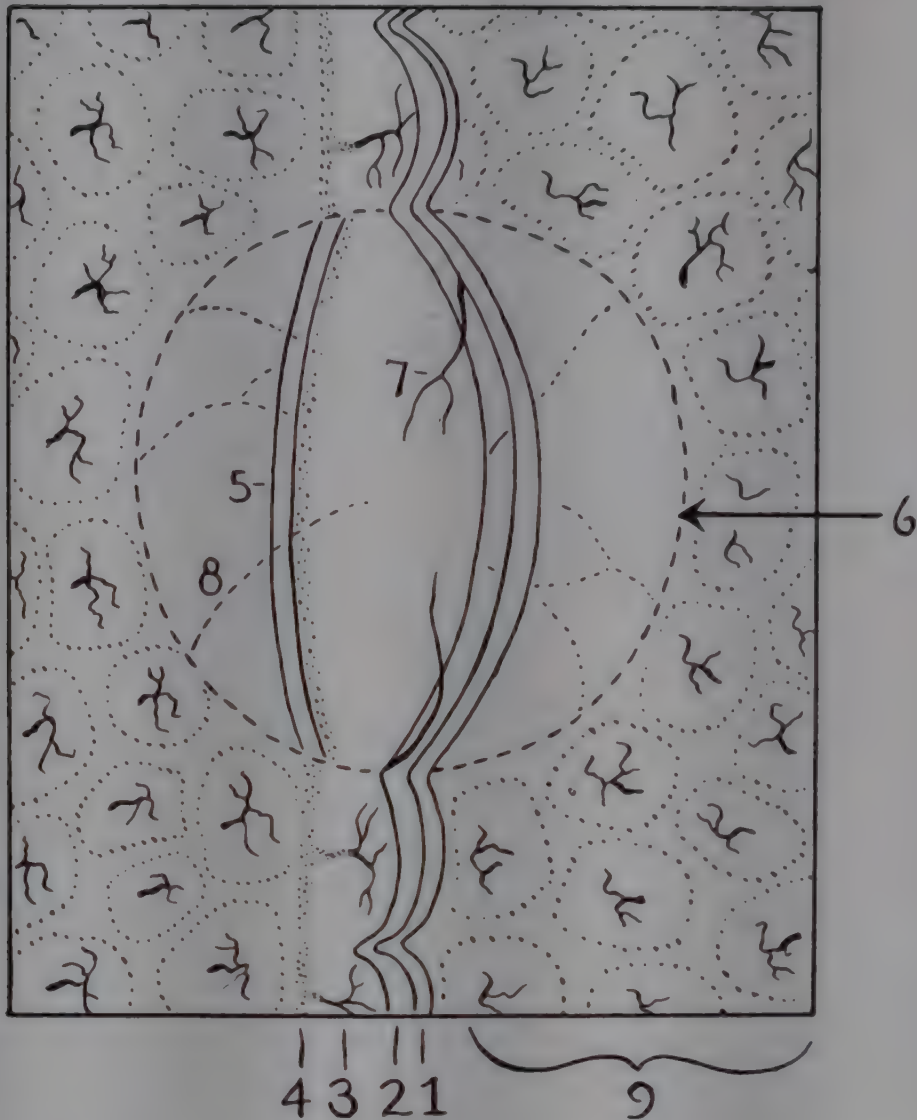


FIG. 124. Diagram showing structure of follicles. 1, Superficial epithelial layer; 2, deep epithelial layer; 3, superficial adenoid layer with vascular buds coming from large vessel (4) in deeper adenoid layer; 5, posterior surface of follicle; 6, outlines of follicle; 7 and 8, superficial vessels over follicle; 9, area showing papillae (extrasectionally).

follicles are invaded by a fibroblastic process leading to the formation of scars. Moreover, they may not only attain a large size but may become confluent.

Except in trachoma,* follicles are most commonly found in the lower eyelid and fornix, where they are arranged in rows, although in patients with lymphatic constitutions the upper eyelid may display follicles near the free border of the tarsus, especially at the lateral angles, the central areas being relatively unaffected.

* The follicle-like excrescences found at the limbus in trachoma are probably the expression of similar reaction.

ACUTE CONJUNCTIVITIS

Hyperemia, edema, secretion, and subconjunctival hemorrhages are common to all forms of acute conjunctivitis. Intense conjunctival edema and secretion create changes in the tissue which diminish its transparency. In acute fulminating cases, small vacuoles and pustules may form; while in the severely toxic types (membranous and diphtheritic conjunctivitis) coagulation necrosis of the superficial epithelial layers may produce a veritable membrane.

Acute conjunctivitis is usually widespread but there are localized types such as phlyctenular conjunctivitis, in which abscess formation is found in the deeper layers of the conjunctiva surrounded by a narrow zone of injection, the adjacent areas being unaffected. In metastatic infections associated with septic embolization similar localized abscess formation may occur.

In the early stages of acute conjunctivitis, biomicroscopic examination of the bulbar conjunctiva reveals marked engorgement of the superficial vessels; by comparison, the deeper ciliary vessels are enlarged to a lesser degree and appear pinker in color (Plate VII, fig. 4). Numerous small extravasations of blood may be seen; capillaries, almost nonexistent before, are now clearly outlined. At the limbus, hitherto invisible, newly engorged capillary loops appear. At the point where the lower eyelid is in contact with the eyeball a tiny lake of lacrimal fluid forms. With instillation of fluorescein a stained film line passes over the surface, touching the eyelid border like the prow of a ship. In acute conjunctivitis, flocculent material is suspended in this lake and causes turbidity (Tyndall phenomenon) analogous to aqueous flare (Plate VIII, fig. 1).

Although the extreme congestion and edema of the tarsal conjunctiva may obstruct the view of the normal vascular architecture, the surface of the tarsal conjunctiva is always dotted with fine red points. When viewed under high power, these points (frequently mistaken with the unaided eye for petechial hemorrhages) are seen to be the terminations of the so-called "bouquets" or vascular tufts (page 178) which develop from the new vessels, growing toward

PLATE VIII

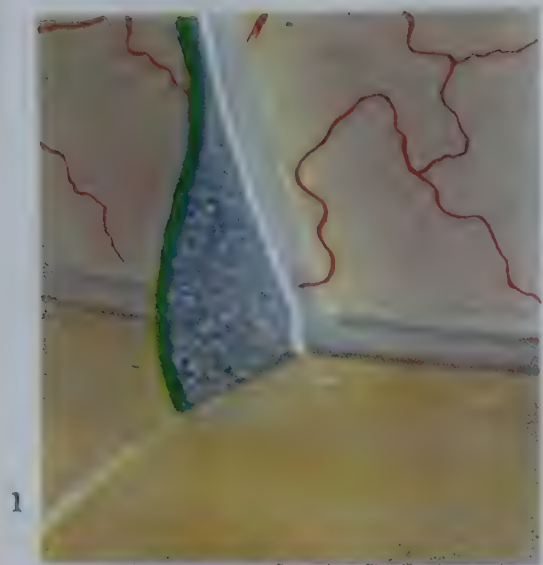
FIG. 1. Catarrhal conjunctivitis with turbidity of the lacrimal fluids beneath the prow line. Direct focal illumination.

FIG. 2. Optic section through phlyctenule stained with azur ii and fluorescein. The central necrotic mass is stained blue with azur and the conjunctival film line is stained green with fluorescein. The periphery of the lesion has an orange tinge.

FIG. 3. Vernal catarrh: palpebral form; a mosaic of flat cobblestone-like excrescences of varying sizes with typical vascularization.

FIG. 4. High power ($40\times$) view of limbal form of vernal catarrh in negro (diffuse illumination).

FIG. 5. Same as Figure 4 in direct focal illumination; note normal (negroid) pericorneal pigmentation.



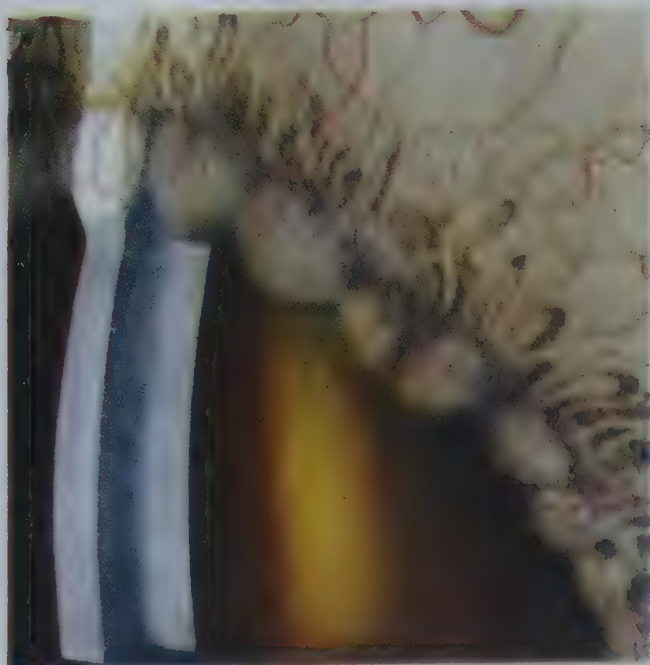
3



4



5



the epithelium. If the conjunctivitis continues, raised papillae will form around these vascular structures. In other words, these "bouquets" not only excite a tissue reaction (infiltration) but also form a skeletal framework for the developing papillae.

SUBACUTE AND CHRONIC CONJUNCTIVITIS

Subacute and chronic conjunctivitides are characterized by formation of papillae and by thickening of the epithelial layer. Considerable mucus is usually found on the surface of the conjunctiva, resulting from the increased secretory activity of the goblet cells. In optic section, the deeper layers of the conjunctiva seem to become irregular, probably due to a downward proliferation of the epithelium. Histologically, proliferation and downgrowth of the epithelium, particularly in the palpebral conjunctiva, form crypts, the adhering walls of which resemble pseudoglands. Exaggeration of the folds and furrows of the tarsal and fornical conjunctiva results in a roughened velvety appearance. In severe conjunctivitis (e.g., gonococcal) these folds may develop into large papilliform excrescences. Genuine hypertrophy with formation of fibrous tissues may result. Concretions may form in these folds or pseudocysts, owing to infiltration with lime. These deposits are seen as small white faceted points, and with the biomicroscope, in optic section, are localized below the epithelium.

In chronic malignant forms, for example, trachoma, xerosis, or pemphigus, keratinization may occur in the exposed areas of the conjunctiva; when this happens the conjunctiva loses its mucous character and resembles skin.

ALLERGIC CONJUNCTIVITIS

In the types of conjunctivitis associated with allergy the conjunctiva, other mucous membranes and skin show like reactions. The causative allergens are usually animal or vegetable substances, but physical irritants and drugs may produce similar irritations. The severity of the reaction in the conjunctiva varies according to the sensitivity of this membrane. When the reaction is transient only

mild hyperemia is observed. Continued hyperemia, however, leads to papillary hypertrophy. An exaggerated form of papillary hypertrophy is found in vernal catarrh. Chronic cases are characterized by formation of follicles, usually on both upper and lower tarsal conjunctivae. When the irritation is caused by atropine, rows of follicles are seen, especially in the lower eyelid, possibly owing to the fact that this region receives the first impact and major effect from the instillation of drops. Although there is still some question as to whether phlyctenular conjunctivitis (eczematosa, scrofulosa, or lymphatica) and vernal conjunctivitis (aestival or spring catarrh, also called phlyctena pallida) should be included in the category of true allergic conjunctivitides, recent evidence indicates that these conditions are due to an allergic response after sensitization of the conjunctiva. The pathogenesis of phlyctenules, according to present-day evidence, lies in tuberculo-allergic hypersensitivity of the conjunctiva.

PHLYCTENULAR CONJUNCTIVITIS

This condition, which usually occurs in children, is characterized by the formation of small isolated infiltrations or nodules; it is thought to be due to tuberculo-allergic hypersensitivity, although phlyctens have been known to occur following sensitivity to such divergent materials as face powder and strawberries. The common site of predilection is the limbus. Because of the frequency of corneal involvement this condition is generally known as phlyctenular keratoconjunctivitis. The corneal lesions may either appear spontaneously or may arise from direct extension of a conjunctival process.* Pathologically, a phlyctenule of the conjunctiva is formed by an aggregation of leukocytes in the deeper layers; the central area of leukocytes may be surrounded by mononuclears and giant cells. Hence, the entire process may be considered as a minute subepithelial abscess (Fig. 125). Typically, conjunctival phlyctenules are surrounded by areas of locally congested vessels, leaving the intermediary conjunctiva unaltered. With the biomicroscope a small solid

* The corneal aspects are discussed on page 481.

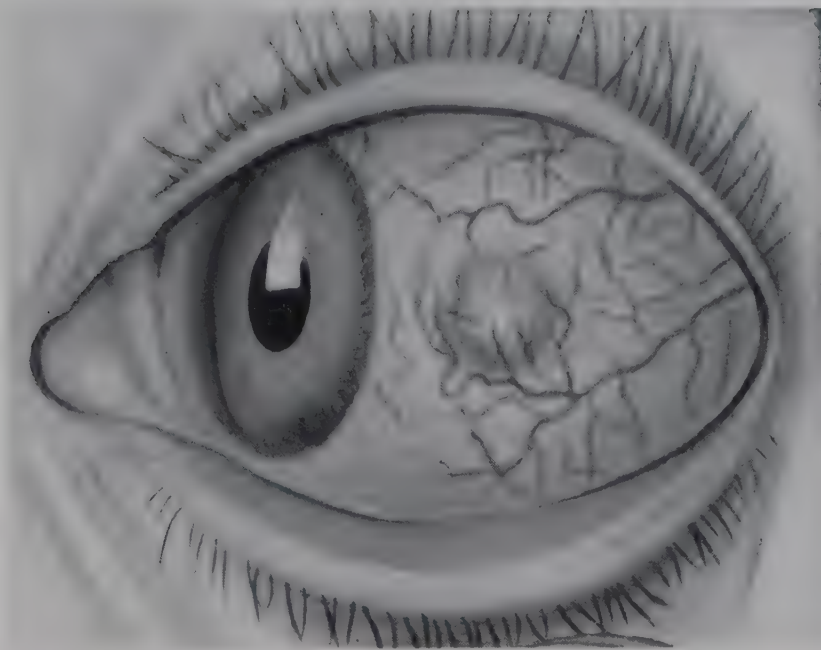


FIG. 125. Solitary phlyctenule of the bulbar conjunctiva.

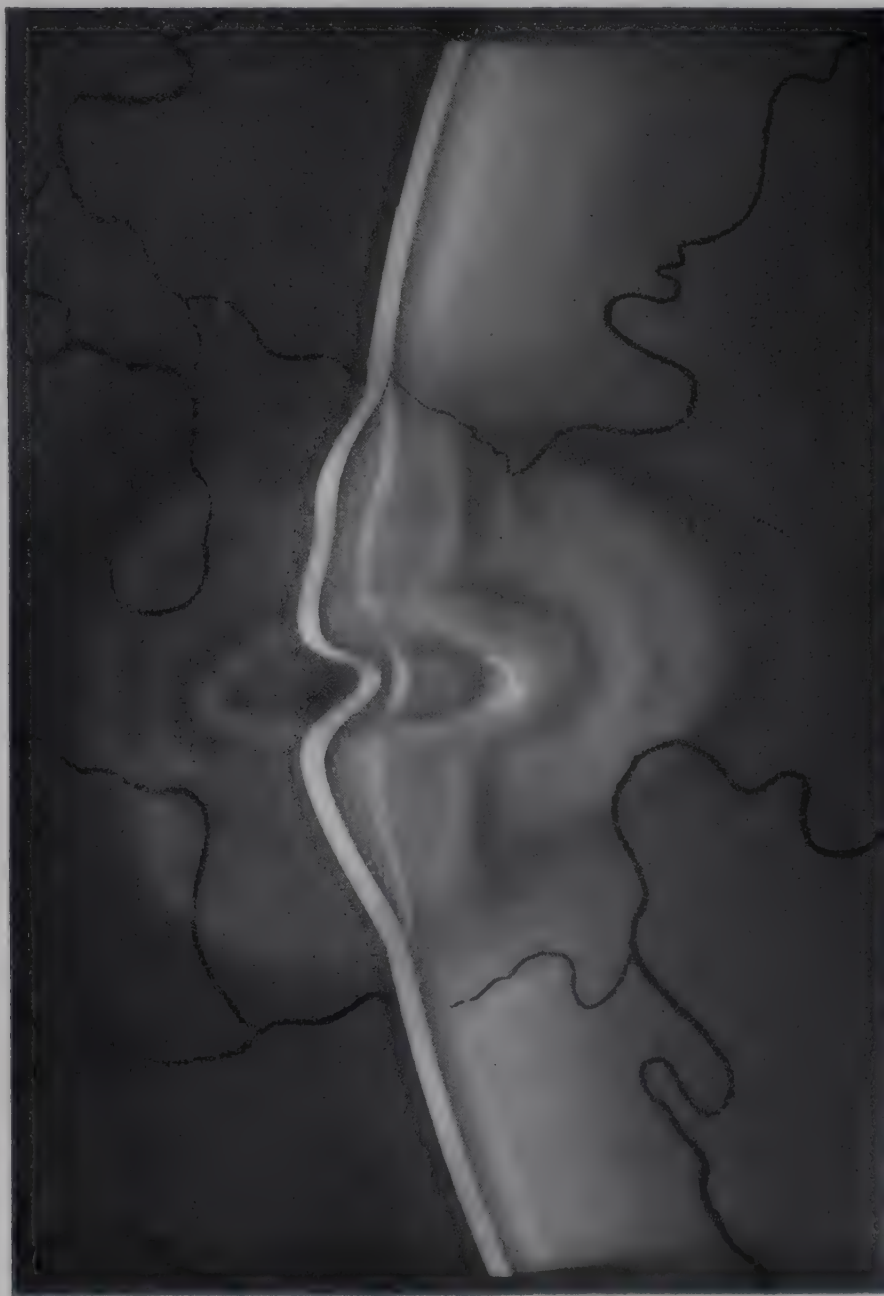


FIG. 126. Optic section through phlyctenule in the bulbar conjunctiva showing central loss of substance.

excrescence is seen, festooned by many dilated capillaries and, unless ulcerated, covered by epithelium. With necrosis, a crater-form central depression appears which stains intensely with azur ii (Plate VIII, fig. 2). When counterstained with fluorescein, the peripheral borders assume an orange hue. In optic section the tear film is seen as a continuous line following the outlines of the phlyctenular border and dipping into the depression (Fig. 126). Depending on the degree of ulceration, healing may occur with or without visible scar formation.

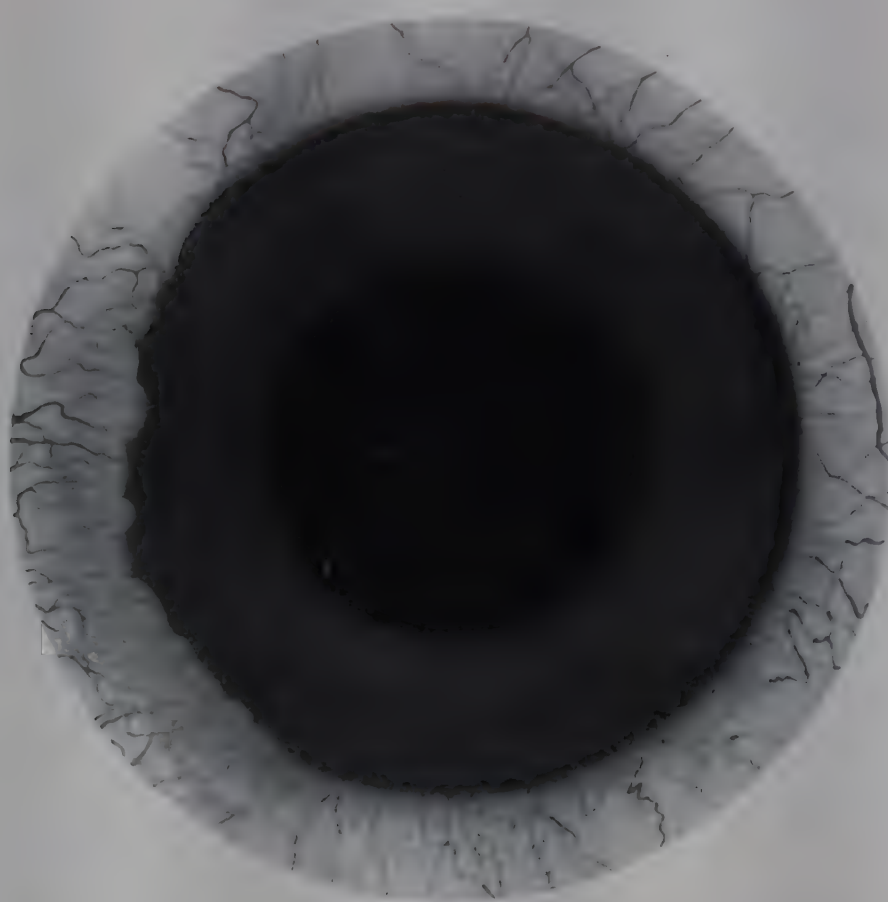


FIG. 127. Vernal catarrh. Limbal form showing gelatinous infiltration extending over to the cornea.

VERNAL CONJUNCTIVITIS (VERNAL CATARRH)

This type of chronic seasonal conjunctivitis usually affects both eyes and occurs in two forms: (1) palpebral and (2) limbal. Both varieties may appear simultaneously.

Palpebral Type. Macroscopically, the tarsal conjunctivae are chiefly involved; their surfaces are covered with numerous, broad and flattened papillae of cartilaginous consistency, producing the so-called cobble-stone or pavement-like appearance (Plate VIII,

fig. 3). These papillae may develop into excrescences of huge proportions. The conjunctiva assumes a bluish white tinge, appears somewhat glazed as though "a thin layer of milk had been poured over it." This appearance is caused by hyaline degeneration of subepithelial connective tissues, which also accounts for the "hardness" of the papillae. In the early stages hyperemia is minimal. In optic section below the epithelium, there is typical proliferation of new vessels (neovascular bouquets), which extend perpendicularly from the deeper parent vessels toward the surface. With the unaided eye they resemble small red points and may be mistaken for petechial hemorrhages. In ordinary papillary formation, each papilla is formed around a *single* vascular tuft, whereas in this condition each large papilla is formed by the confluence of two or more smaller ones. A secondary, more superficial arborization, which forms a reticulum typical of this condition, emerges from the primary tufts. A thick, stringy secretion, usually found on the surface and between the papillae, can be removed without causing bleeding.

Bulbar Type. Small grayish gelatinous elevations seen at the limbal region are arranged concentrically to the cornea, especially in the exposed intrapalpebral portions (Fig. 127). Capillaries from the limbal arcades invade the growth. These elevations occur singly or may coalesce; in the latter case a raised ring is formed, partly or completely encircling the cornea. Almost invariably small subepithelial dots are seen in the adjacent cornea (Fig. 128). As the process develops, the gelatinous ring may encroach on the cornea. However, the major portion of the infiltra-



FIG. 128. Vernal catarrh. Small subepithelial dots seen in neighboring cornea by optic section, 40 \times . Same case as shown in Figure 127.

tion involves the bulbar conjunctiva. In direct focal illumination the gelatinous mass is faintly translucent (Plate VIII, figs. 4, 5). Small whitish spots, described by Trantas, have been noted embedded in the gelatinous mass. Cystic degenerations have been observed by Pascheff²⁸³ and Gabardi.¹¹²

Abortive Vernal Conjunctivitis. An abortive or transient form of vernal catarrh has been observed; the tarsal papillae are small and a pale reddening (mild hyperemia) of the bulbar conjunctiva occurs. This type may be difficult to diagnose except for the bluish white appearance of the conjunctiva, the history of seasonal occurrence, and the subjective symptoms (little or no secretion but marked lacrimation and itching).

FOLLICULAR CONJUNCTIVITIS

As previously stated, papillae are the *vascular* response to irritation in the tarsal conjunctiva, while follicles are the nonspecific response to irritation of the adenoid tissue. Follicles usually appear in the palpebral conjunctiva; less frequently in the fornices and in the region of the caruncle (Plate IX, fig. 1). In the early stages there is no difference either macroscopically or microscopically between the follicles of initial trachoma and those of the follicular conjunctivitis. Consequently, a differential diagnosis cannot be made from the presence of follicles alone. In early cases a more reliable diagnostic clue is their location, since in trachoma the upper eyelid is the site of predilection, whereas in the follicular conjunctivitis the lower eyelid is usually involved to a greater extent. However, in early trachoma, careful biomicroscopic inspection of the limbal regions will almost always reveal corneal involvement, that is, follicles and vascularization (pannus).

The follicular conjunctivitis may appear in either acute or chronic forms. In one acute form (Beal), there is rapid onset accompanied by a preauricular adenitis. The clinical symptoms being relatively mild, complete resolution in a few weeks is the rule. The tarsal conjunctiva, especially of the lower eyelid, becomes markedly engorged. Although some papillary hypertrophy may occur it is soon

overshadowed by extensive follicle formation. The bulbar conjunctiva is injected, particularly in the region of the fornices. There is only a small amount of thin serous discharge. In its early state, this type of conjunctivitis may easily be confused with acute trachoma.

The chronic form of follicular conjunctivitis is a benign disease, characterized mainly by a concentration of small discrete follicles in the lower fornix. Complete resolution is the rule although the course may be protracted; however, such sequelae as scarring or corneal complications never occur. With the biomicroscope rows of densely packed, variously sized follicles are seen, their characteristic structure and vascularization being discerned in optic section.

VIRAL CONJUNCTIVITIS

EPIDEMIC KERATOCONJUNCTIVITIS

In 1939 zur Nedden³⁴³ reported 200 cases of a rapidly spreading form of acute conjunctivitis associated with corneal lesions, which occurred in Düsseldorf. In 1938 Schneider²⁶⁹ had published an account of 150 cases of this disease which he had seen in Munich. Two years later an epidemic occurred in Oahu, Hawaii, and was reported by Holmes.¹⁵² Accounts of similar epidemics have come from India, Tasmania and recently from the western coast of the United States.

The German writers emphasized the epidemic nature of the condition, the severe conjunctivitis, the corneal lesions which often could be seen only with the biomicroscope and the presence of adenopathy. They called the condition keratoconjunctivitis epidemica.

Epidemic keratoconjunctivitis is characterized by sudden onset, with pain, like that caused by a foreign body in the eye, excessive lacrimation, and marked edema. On the second or third day swelling of the preauricular node occurs; this may or may not be painful and generally subsides within a week. Although in the first few days secretion is minimal, later it may become profuse and membranes, dirty gray in appearance, may form. Removal of these mem-

branes may leave raw bleeding points. In about half the cases the fellow eye becomes involved after the first week (Figs. 129, 130). Extreme chemoses of the lower bulbar conjunctiva, so characteristic



FIG. 129. Epidemic keratoconjunctivitis. Diffuse illumination; early folliculosis of lower eyelid.

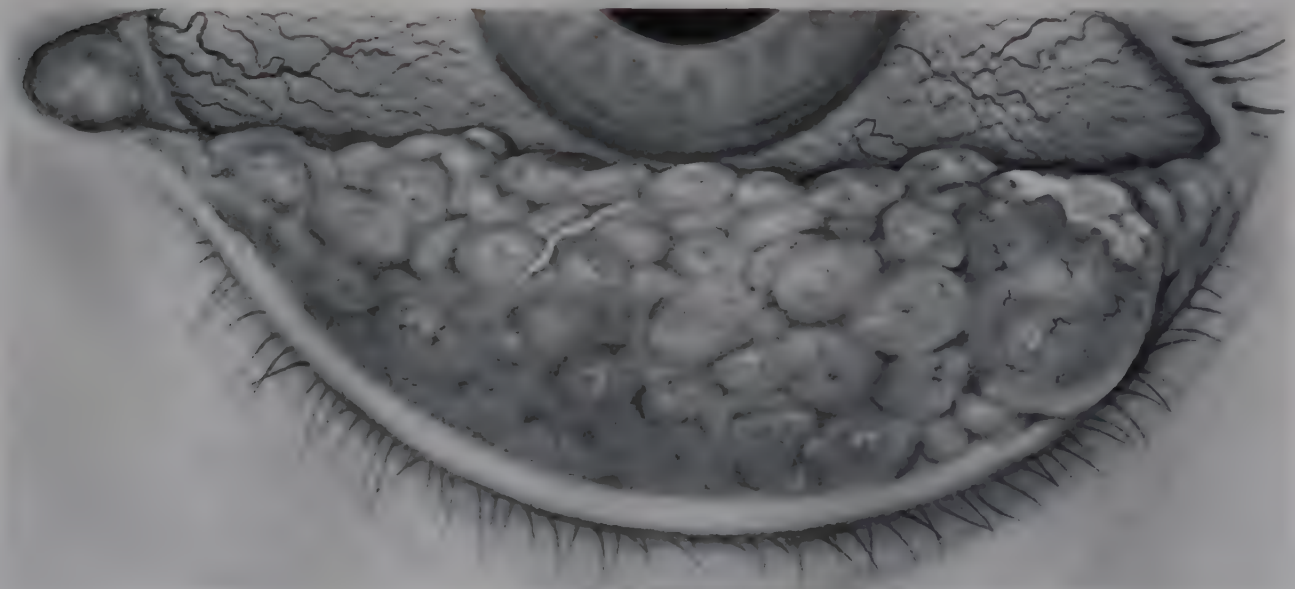


FIG. 130. Epidemic keratoconjunctivitis. Diffuse illumination; later stage of folliculosis with exudative membranes.

of this condition, may cause protrusion of this membrane over the lid margin.

As a rule the acute symptoms begin to abate at the end of the second or the beginning of the third week. The swelling and secretion diminish leaving a thickened and reddened mucosa, which persists owing to residual folliculosis. This may continue for from four

to eight weeks before complete resolution occurs. About the time the acute symptoms subside more than half the patients complain of blurring of vision. This results from the presence of small dis-

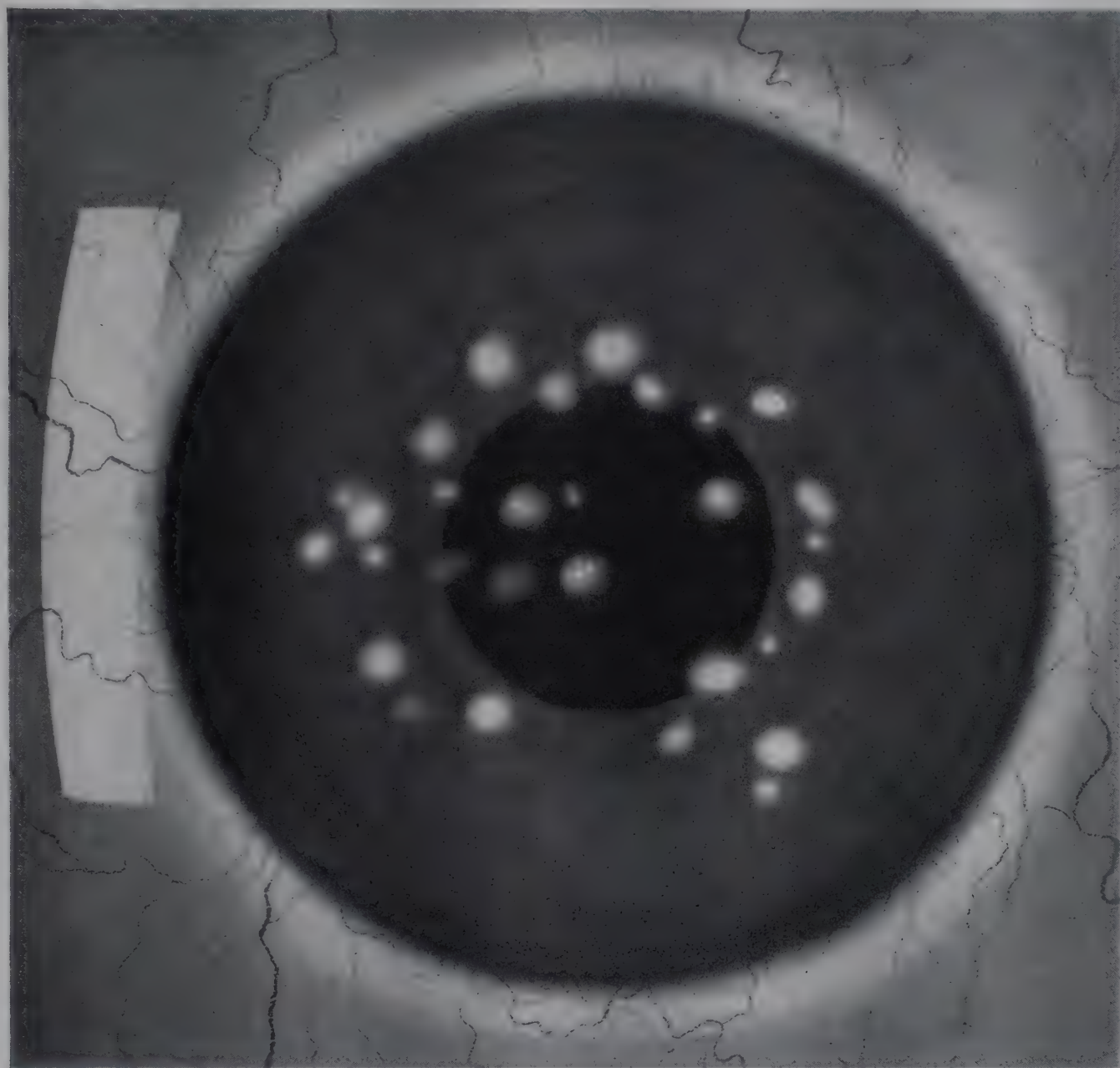


FIG. 131. Epidemic keratoconjunctivitis. Corneal opacities by sclerotic scatter.

crete grayish infiltrates which occupy the pupillary area and are located in the basal layer of the epithelial cells or in Bowman's zone (Figs. 131, 132, 133).

Beginning late in December, 1941, I had occasion to see eighteen persons having this type of conjunctivitis. In nine instances the infection spread to the opposite eye and in fourteen corneal opacities appeared, reducing the vision on an average to about 20/50. As time went on the vision gradually improved and in most instances

three months after the onset the spots became less opaque and in ten cases the vision improved to 20/30 or to 20/20 -. In four cases the spots were very small and no evidence of their presence could



FIG. 132



FIG. 133

FIG. 132. Epidemic keratoconjunctivitis. Corneal opacity in parallelepiped (high power).

FIG. 133. Epidemic keratoconjunctivitis. Location of corneal opacities in optic section.

be seen after three months. In three cases the corneal spots were seen on the third day following the onset of the conjunctival congestion. In one case these lesions assumed polymorphous forms rather than the usual circular shape, appearing as short, irregular lines.

By retro-illumination the corneal surface resembled that seen in

mild keratoconjunctivitis sicca, although no filaments were present. In the early stages the corneal lesions can be stained with fluorescein; then they frequently have a pyriform shape, the tapering greenish stained ends coming to the surface of the tear film line. Later the connections with the surface epithelium become lost and after two or three weeks they are found in the deepest (basal) epithelial layer as circular and granular lesions. At times they appear to lie between the epithelium and Bowman's zone. In this later stage several unsuccessful attempts were made to stain them by repeated instillations of fluorescein over a period of twenty minutes.

Biomicroscopic examination of the conjunctiva during the height of the acute stage revealed rows of closely packed hemispheric follicles interspersed with occasional papillary formations. This was particularly marked in the lower lid and cul-de-sac. The bulbar conjunctiva was injected and chemotic. In the subacute stage the follicles gradually diminished in size but did not completely disappear until two months after their initial appearance. Likewise, the small discrete corneal opacities which were made up of fine whitish granular dots became grayer and thinner as time went on, and in four instances they could no longer be seen after three months.

Although this condition bears similarity to Béal's type of conjunctivitis it differs in that it is much more severe and has corneal complications.

Bacteriologic studies by separate workers have uniformly proved negative. The general feeling is that the condition is due to a virus infection. Thygeson says in a report: "The cases which we have seen, including the one you sent up, have shown negative bacteriological findings, but all have shown a mononuclear cell exudate. Two of the cases have suggestive basophilic inclusions in the cytoplasm of epithelial cells, but this finding has not been consistent enough to be significant. There has been no transmission to any of the ordinary laboratory animals, but there have been a few apparent successful transmissions intracerebrally in mice producing meningoencephalitis, but it has not been possible to maintain any of these in series. Doctor Sanders is now attempting to obtain a strain

which can be maintained in mouse and tissue culture, but has not as yet been successful. I feel that the results, though negative, definitely suggest a virus etiology."

INCLUSION BLENNORRHEA (NEONATORUM)

An acute type of follicular conjunctivitis, attributed to a virus infection because of the presence of cellular inclusions, occurs in the newborn. Clinically this must be differentiated from the mild form of gonococcal ophthalmia neonatorum. After the initial acute conjunctivitis (hyperemia) has subsided somewhat, the subacute and chronic stages are marked by the presence of follicles. The condition is usually unilateral and resolves completely without untoward sequelae. However, rarely and in severe cases fine scarring has been observed in the conjunctiva. Biomicroscopic studies of the eyes of the newborn cannot obviously be easily made.

SWIMMING-BATH CONJUNCTIVITIS

This is an analogous type of virus inclusion conjunctivitis which is found among the older age groups. It is generally attributed to infection incurred after bathing in a swimming pool. The acute stage is characterized by hyperemia and congestion. The bulbar conjunctiva, if involved at all, shows only a slight infection. After one week or more, the acute symptoms subside; then follicles slowly appear in the thickened tarsal conjunctiva. The folliculosis may last for several months before disappearing but there are no permanent sequelae.

HERPES ZOSTER OPHTHALMICUS

Involvement of the conjunctiva by herpes zoster is comparatively rare. Occasionally, in association with cutaneous involvement of the eyelids, there is an isolated lesion of the bulbar conjunctiva. It appears as a phlyctenule-like raised nodule, which soon tends to soften and ulcerate in the center. The nodule is surrounded by considerable conjunctival injection. The lesion heals by cicatriza-

tion, leaving a round scar, which may be dark in color, owing to deposition of melanin or to thinning of the subconjunctival tissues (Fig. 134).

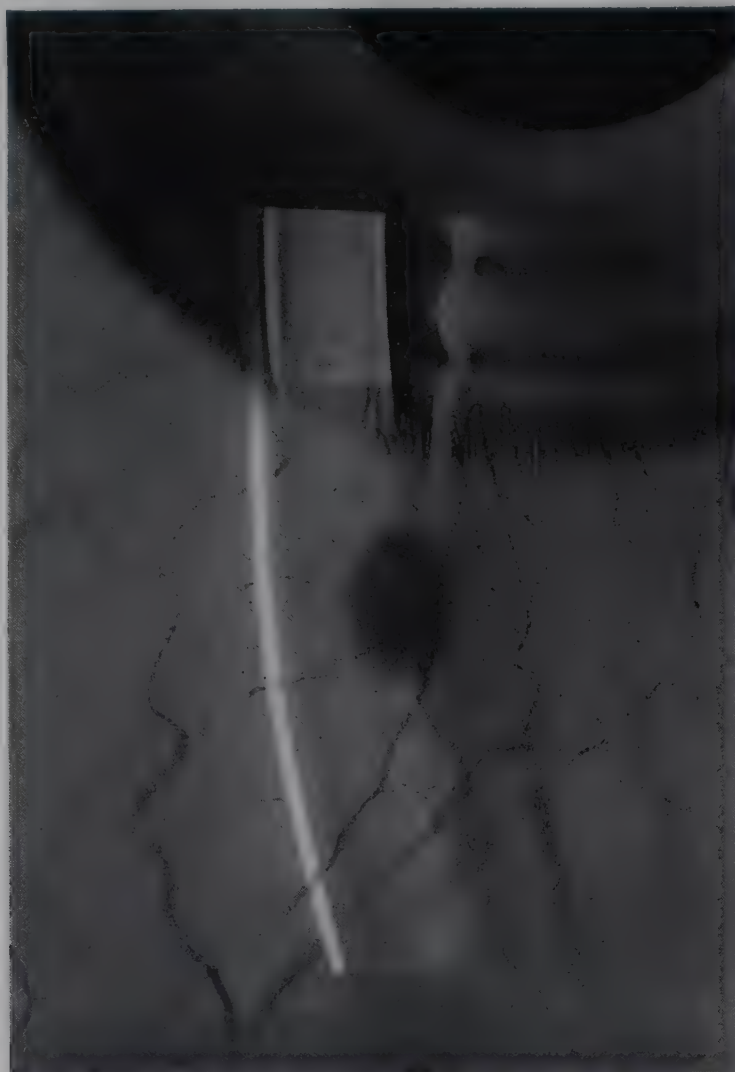


FIG. 134. Herpes zoster ophthalmicus — healed stage. Dark round scar is seen in the deeper conjunctiva near lower limbus.

TRACHOMA

This disease is of especial importance to the biomicroscopist. According to many writers biomicroscopic examination of the conjunctiva is indispensable to early diagnosis of trachoma. Since the etiologic agent is still unknown — although because of the presence of cell-inclusions, virus infection is suspected * — the diagnosis of trachoma depends in part on its clinical aspects. From this standpoint, trachoma is to be considered as a form of keratoconjunctivitis, characterized by chronic inflammation of the epithelial and sub-

* The most recent evidence advanced by Cuénod and Nataf suggests that it is a rickettsiosis. However, both Julianelle and Thygeson have been unable to verify this finding.

epithelial layers. It is marked by papillary hypertrophy, extreme lymphoid hyperplasia with follicle development, pannus and eventual cicatrization. Even at the onset, when, with the naked eye, only a mild degree of congestion is seen, biomicroscopic examination may reveal features which, although not yet differentiated, are already typical of this disease.

Cuénod and Nataf have given an excellent biomicroscopic analysis of trachoma, using the four-stage classification of MacCallan. They fortunately were able to examine early cases in children having "pure" trachoma, and thus could eliminate changes resulting from contamination.

TRACHOMA I

Except for a pinkish blush, the conjunctiva, in the early stages of infection, may appear normal to the unaided eye. However, biomicroscopic examination of the tarsal conjunctiva will demonstrate the presence of small reddish dots, immediately beneath the epithelium, which, when seen in optic section and with high magnification, are distinguished as the newly formed perpendicular vessels (incipient stage of papillae formation) (page 178). At the same time small, round, slightly protuberant pale areas, which are the earliest manifestations of the developing trachomatous follicles, are discerned among the budding papillae (Plate IX, fig. 2). As the condition progresses the deeper vascular elements become veiled by the thickened mucosa, only the larger vessels at the free margin of the tarsus being still in evidence.

A coexisting infiltrative haze at the limbus and proliferation of the superior limbal arcades over the cornea are always seen in the early stages of trachoma. This signifies that the cornea is the site of specific primary involvement at the very outset of the disease.

TRACHOMA II

The velvety appearance of the entire conjunctiva owing to the progressive papillary hypertrophy, is the most striking feature of this stage, which is characterized by the development of papillae and

follicles. The superficial manifestation of these papillae is demonstrated, especially by vital staining, as a mosaic-like pattern in the conjunctiva. Each tile of the mosaic represents one papilla. Simple papillary hypertrophy, as seen in most types of benign conjunctivitis, resembles this lesion, but the combination of papillary hypertrophy, follicle formation and corneal involvement, as seen in trachoma, is distinctive. The follicles protrude between the papillae as if pushing them aside. Follicles may also appear on the caruncle and in the semilunar fold. Aubaret attributes to this fact great diagnostic importance. The follicles are usually largest on the tarsal conjunctiva and in the fornices; they are from four to six times as large as the papillae and appear as translucent, hemispheric protuberances (Plate IX, fig. 3). In optic section the contents look structureless. At the superior tarsal margin, the follicles have a milky appearance, are increased in size, and are frequently confluent. In contrast with the papillae which have central budding vessels, the follicles have a special type of vascularization. With high magnification, small capillaries may be seen climbing over their surfaces and frequently actually penetrate into the follicles. High magnification after vital staining with azur ii brings certain features into beautiful relief, exhibiting follicles of a light bluish tinge and papillae outlined at their periphery by fine dark blue dots, the whole resembling a hexagonal mosaic. By diaphanoscopy (Trantas) papillae can be easily differentiated from follicles. The former have a solid meaty appearance, while the latter resemble opalescent globes.

In addition to the papillary and follicular changes already noted, larger cystlike formations occur, especially on the upper conjunctiva and caruncle. These burst easily on manipulation, exuding their clear gelatinous contents. They are bleblike and resemble small "sago grains" but may become larger, blending with the thickened, swollen conjunctiva and producing a gelatinous appearance of the free tarsal border. They result from proliferative epithelial down-growth into the submucosa with the formation of pseudoglands. Cystic degeneration of strangulated conjunctival and tarsal glands accounts for the muroid contents of these structures.

PLATE IX

FIG. 1. Follicular conjunctivitis of the tarsal conjunctiva. Dense carpet of follicles with their characteristic vascularization.

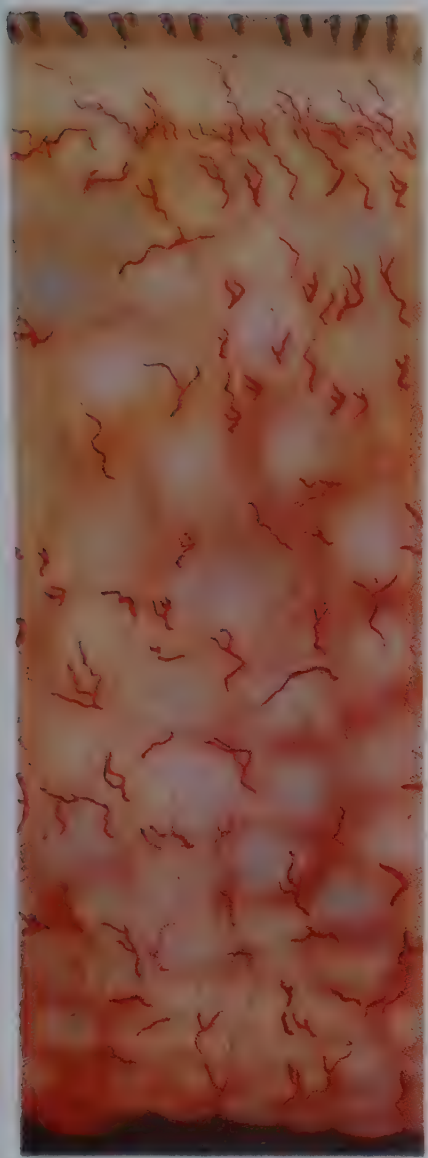
FIG. 2. Trachoma I. Follicles and early stage of papillary formations. Upper tarsal conjunctiva.

FIG. 3. Trachoma II. Optic section through tarsal conjunctiva. Papillae and one large follicle (60 \times).

FIG. 4. Trachoma, stage III (everted upper eyelid). Stellate scars and absorbing follicles in diffuse illumination.

FIG. 5. Optic section through a follicle and stellate scars. Same case as shown in Figure 4. Film line stained with fluorescein. 40 \times .

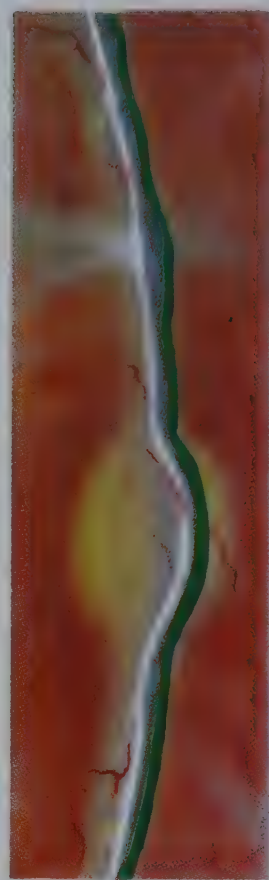
FIG. 6. Trachoma IV. Scar tissue (Arlt's line).



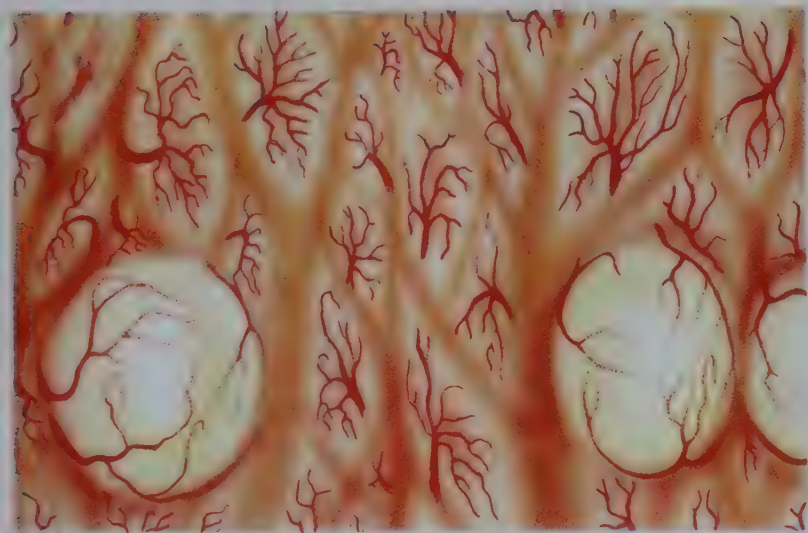
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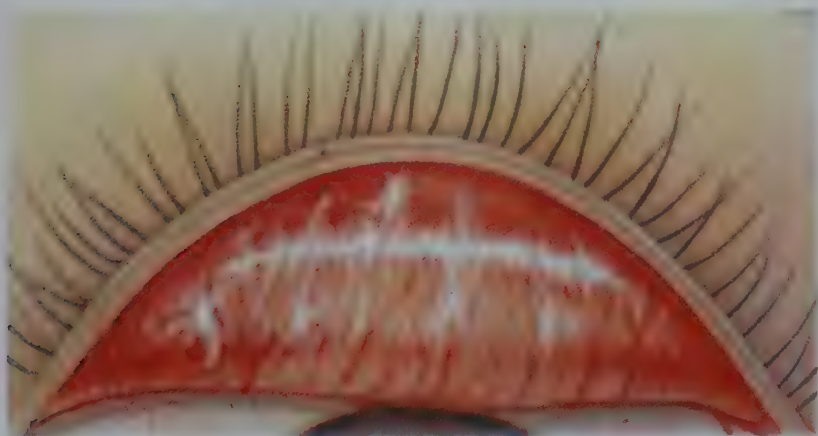
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TRACHOMA III

The healing stage begins with the formation of fine cicatricial bands which appear as delicate white lines between the papillae. The follicles which are in the process of absorption or have ruptured or become necrotic are slowly invaded by scar tissue, producing *stellate* white scars at their sites. The biomicroscope reveals these stellate scars long before they are visible to the naked eye (Plate IX, figs. 4, 5).

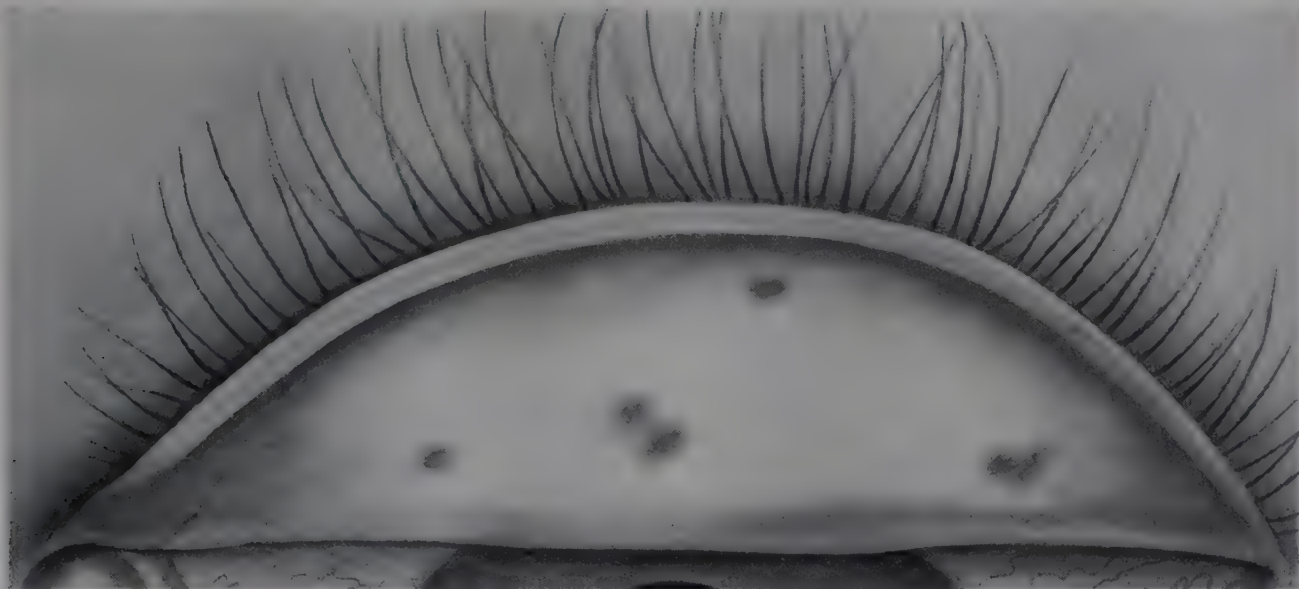
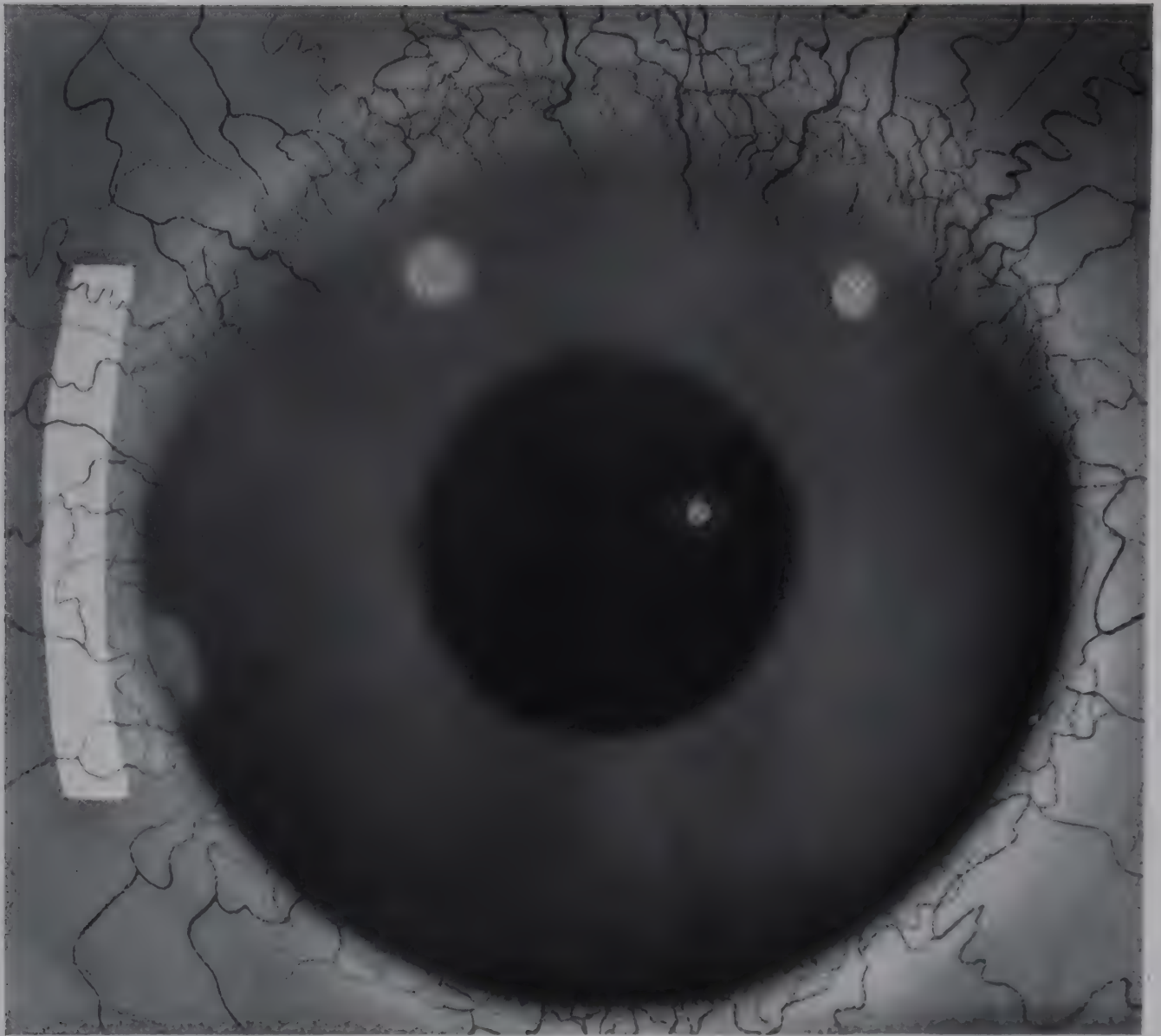


FIG. 135. Trachoma IV. Marked cicatrization of upper tarsal conjunctiva.

TRACHOMA IV

This stage is characterized by a progressive increase in cicatricial tissue which leads to the obliteration of the follicles and papillae, resulting in an avascular smooth white surface. After the formation of the early cicatricial stellate scars, the cicatricial line of Arlt develops (Plate IX, fig. 6). This is a horizontal white band of scar tissue in the middle of the tarsus in the zone of the anastomosing vessels. As cicatrization progresses, small red areas of vascularized papillae may remain, but in the final stage of this process the conjunctiva assumes a glistening white appearance, devoid not only of papillae and follicles but also of all vascularization (Fig. 135). Contraction of the scar tissue leads to the dire sequelae of this disease, which are due mainly to deformity of the eyelid or to dryness



A



B

FIG. 136. A. Trachomatous pannus. Follicular scars somewhat removed from the limbus. Sclerotic scatter. Early case of trachoma. B. High power view of the nodes or follicular scars seen in Figure 136 A.



C



D

FIG. 136. c. Same node in optic section (high power view). d. Follicle (rosette) at the limbus (40 X), early stage.

of the eye following destruction of the secretory elements. Entropion, trichiasis, symblepharon are associated with the former; xerosis, madarosis (loss of cilia), and ptosis follow with the latter. Further, the accompanying pannus leads to loss of vision.



FIG. 137. A. Diffuse type of pannus in a case of trachoma of five years' duration (sclerotic scatter.)

PANNUS TRACHOMATOSA

From biomicroscopic studies, it is now known that corneal involvement (pannus) is not a secondary complication of trachoma, but is a specific part of the disease, appearing early in its course. The corneal lesion, which appears almost immediately after the onset of the disease as an alteration of the limbal relucence and as vascularization, may be so delicate as to be overlooked by ordinary methods of examination. The importance of biomicroscopic examination

of the upper limbal areas at this stage should be strongly emphasized (Fig. 136, A, B, C, D).

Observation of the upper limbal region even with low power



FIG. 137. B. Optic section view of case shown in Figure 137 A, nasal side of cornea. Illustrates location of superficial scars and vessels.

shows mild congestion of the small vessels of the limbal network, from which newly formed capillary loops extend superficially into Bowman's zone. With high power and thin optic section, the corneal tissue surrounding and preceding these subepithelial capillary loops appears slightly relucet. By retro-illumination or sclerotic scatter a delicate haze will be seen. In these areas lying at a level with Bowman's membrane, the opacity is caused by many grayish points and lines. Within this area of grayish infiltration, small follicles may appear as slightly raised, translucent nodules. Some of these follicles when surrounded by a fine capillary network form structures

known as Herbert's rosettes. This follicular formation corresponds to that of the conjunctiva, modified by the peculiar anatomy of the cornea (Fig. 137). Follicles may be abortive or may have a complete

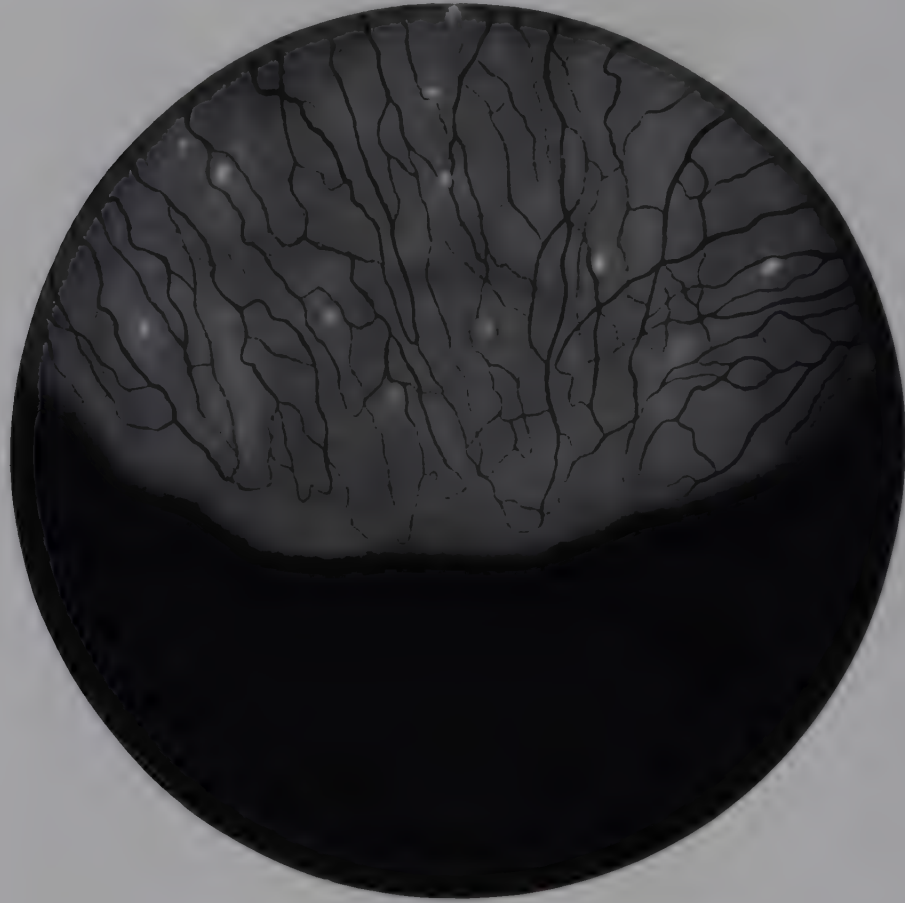


FIG. 138. Pannus trachomatosa (crassus).

life cycle; after reaching maturity they empty and organize, leaving small opaque scars or nodes covered by epithelium. In some instances clearer depressions may result. These depressions are likely to occur near the limbus and are known as Herbert's marginal pits (Fig. 139). In addition, small superficial subepithelial infiltrations may break down and subsequently heal by epithelialization and thus cause irregularities (facets) of the corneal surface. The capillary loops progress downward toward the pupillary area, terminating in a more or less horizontal line which is always preceded by a grayish area of faint infiltration, containing clear streaks. These clear streaks may serve as preformed avenues for the extension of the capillary loops. As pannus regresses, these areas are the first to clear. At first, the infiltration of pannus is superficial; but in severe cases, after destruction of Bowman's membrane, the process may infiltrate

into the deeper layers, resulting in deep scarring and vascularization with irregular thinning of the entire corneal thickness. This occurred in about 50 per cent of the cases seen in our clinic. Sec-



FIG. 139. Herbert's pit at limbus, 40 \times .

ondary degenerative changes such as calcareous deposits, fatty and hyalin degeneration, and keratectasia are found with pannus of long duration. Although pannus formation tends to advance from the upper limbus downwards, there are instances when the process develops in other sectors (Fig. 137A), or even circumferentially. Variations in appearance or development of a pannus probably depends not only upon the time element or on the intensity of involvement, but also on the resistance of the involved tissue. Further, many of its aspects may be varied or modified by therapy. When thin it is termed *pannus tennus* (Plate XXVII, fig. 1), when highly vascular *pannus vasculosus* and when thick or hypertrophic *pannus crassus* (Fig. 138). As mentioned under the heading of Trachoma IV (p. 207) the secondary changes in the cornea and conjunctiva (scarring and destruction of the secretory elements), may result in a picture not dissimilar to that occurring in pemphigus (Plate XI, figs. 1 and 2).

TUBERCLE OF THE CONJUNCTIVA

Although many cases of tubercle of the conjunctiva have been described in the literature, this condition is so rarely seen in clinical

practice that there are few reports of biomicroscopic examination. Because of the morphologic nonspecificity and the large size of the lesions, biomicroscopy has not been of great service in differential diagnosis.

Basing his study on the work of Sattler,²⁰⁷ Eyre⁸² described four types of lesions: (1) The ulcerative, characterized by small miliary ulcers which are chronic and may spread to the cornea with pannus formation, causing considerable destruction. (2) The nodular type, characterized by subconjunctival nodular elevations, which at first are small, grayish yellow in color, and at times resemble the follicles of trachoma. As they increase in size, owing to the development of granulation tissue, large conjunctival masses form, some having a central area of ulceration (Plate X, figs. 1, 2, 3, 4). (3) The papillary hypertrophy type, sequela of the nodular type. Large jelly-like masses of granulation tissue appear on the tarsal and fornical conjunctivae; they have a tendency to become pedunculated. (4) The polypoid type, exemplified by small pedunculated fibrous tumors. A metastatic form, described by Lafon¹⁸⁷ and Junius,¹⁶² is a conjunctival tuberculoma, which appears as a firm, solid, reddish yellow subconjunctival tumor of the bulbar conjunctiva, unassociated with inflammation.

In lupus erythematosus, extension to the conjunctiva may occur from the neighboring skin lesions in the form of a chalazion-like nodules of an evanescent nature, situated on the bulbar conjunctiva. These lesions do not break down but tend to atrophy and result in cicatrization. At the same time, the bulbar conjunctiva may exhibit intense congestion with either localized or diffuse small grayish infiltrative patches. These sometimes appear as small rounded lentil-like gelatinoid masses. The process may extend over the surface of the cornea either superficially or deeply as an interstitial keratitis. (See tuberculosis of the cornea, page 508.)

Rarely tuberculides of the conjunctiva appear. The most frequent form is that known as lichen scrofulosus, which consists of small nodules of an evanescent nature, situated on the bulbar conjunctiva.

SYPHILIS OF THE CONJUNCTIVA

Syphilitic lesions of the conjunctiva, like those of other exposed mucous membranes, present numerous manifestations. The type and

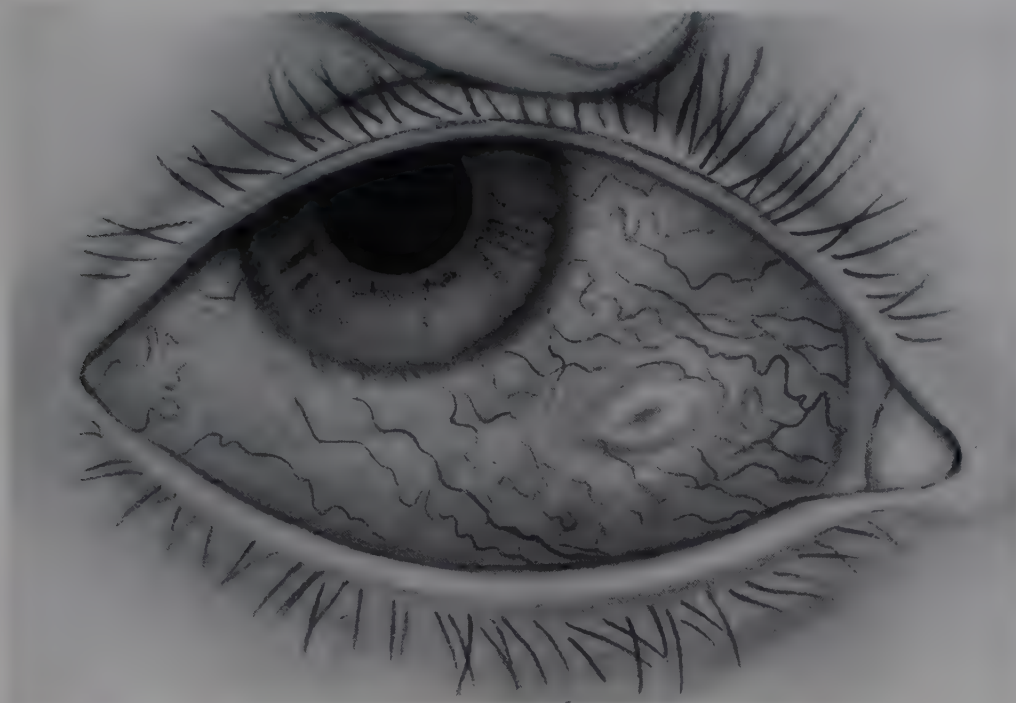


FIG. 140. Syphilis. Primary chancre of the conjunctiva. (After W. Clausen.)

appearance of the lesion varies with and is governed by the stage of the disease.

The conjunctiva, in comparison with other mucous membranes, is a not infrequent site of extragenital chancre. Although involvement of the bulbar conjunctiva and upper eyelid are most common, chancres of the lower tarsal conjunctiva and the caruncle have been reported (Fig. 140; Plate XI, fig. 6). The lesion starts as a hard, indolent, painless papule, which soon develops into a typically "punched-out," spreading ulcer with an indurated base and a necrotic center.

In the secondary stages the disease may be manifested by simple conjunctivitis of varying severity, characterized by ordinary papillary hypertrophy and edema, or by a granular conjunctivitis (pseudotrachoma or conjunctivitis papulosa). There has been considerable controversy concerning granular syphilitic conjunctivitis, because of its resemblance to trachoma. The former is characterized

PLATE X

FIG. 1. Tuberculosis of the conjunctiva. General view of the everted eyelids. Early nodular type. (After Renard and Nataf.)

FIG. 2. Enlarged view of lesion shown in Figure 1. (After Renard and Nataf.)

FIG. 3. Tuberculosis of conjunctiva. Later stages of nodular type. Lesions tend to have necrotic centers. (After Renard and Nataf.)

FIG. 4. Enlarged view of lesions shown in Figure 3. (After Renard and Nataf.)

FIG. 5. Parinaud's conjunctivitis. Follicles of varying sizes.

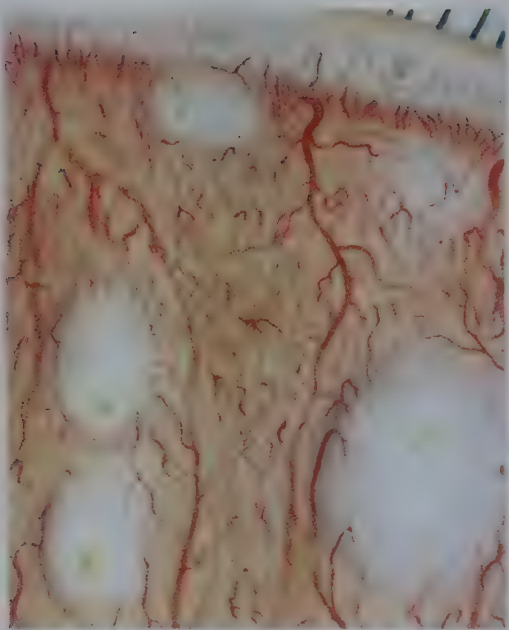
FIG. 6. Parinaud's oculoglandular syndrome; vegetative form. Enlarged view of conjunctival vegetations. Large papillary formations and follicles. (After Renard and Nataf.)



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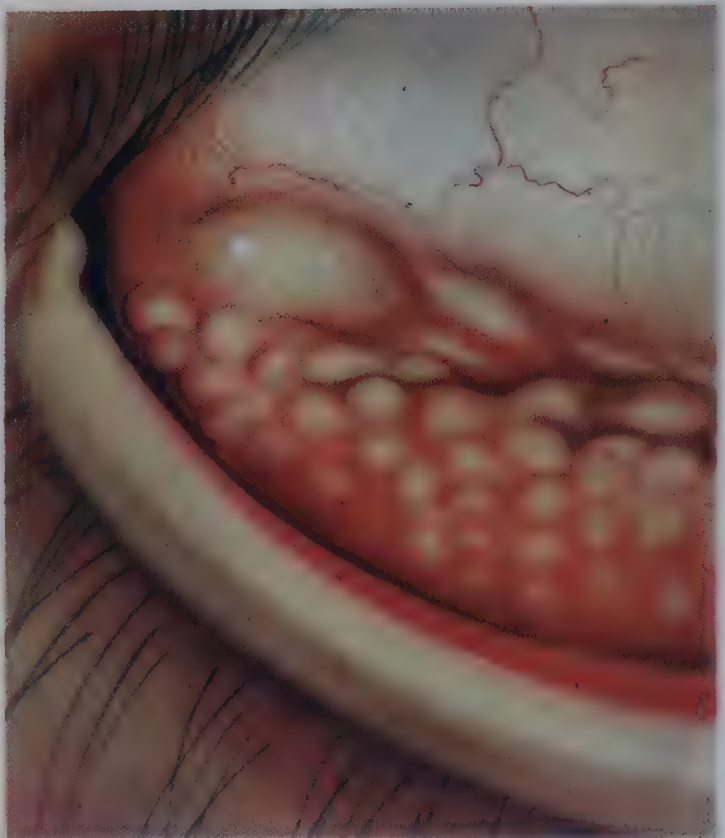


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by thickening and papillary hypertrophy of the tarsal conjunctiva and occasionally by accompanying exuberant pannus. Although the histologic studies of Igersheimer have shown that the diffuse follicular-like growth on the tarsal conjunctiva is due to a simple papillary hypertrophy rather than to real follicle formation, there have been no biomicroscopic observations to confirm this point of view. Clinically, the absence of follicles would tend to rule out trachoma.

Syphilids of the conjunctiva are rare but when they do occur, they appear as small, grayish, elevated patches surrounded by a narrow zone of injection. They may be situated on either the bulbar or tarsal conjunctiva or caruncle; they have a phlyctenule-like appearance or, less commonly, that of exuberant, jelly-like, pinkish masses.

The tertiary manifestations of syphilis are exemplified by gummas of the conjunctiva. They usually appear on the bulbar conjunctiva at the limbus, as small, hard, pinkish nodules which later may ulcerate. In untreated cases the cornea and sclera may be involved, leading to such unfortunate sequelae as iritis, and even perforation with panophthalmitis. When gummas occur on the tarsal conjunctiva, extension through the deeper eyelid tissues may result in an orbital cellulitis with exophthalmos.

Under specific antisyphilitic treatment, the lesions, in their early stages, resolve rapidly, and the serious sequelae do not appear.

LEPROSY OF THE CONJUNCTIVA

There have been only a few accurate biomicroscopic descriptions of conjunctival leprosy. According to Pinkerton²⁴² pure leprosy involvement of the conjunctiva is rare. Barros²² in a study of 1200 cases confirms this opinion. He believes that the bulbar conjunctiva is chiefly affected by the spreading of episcleral lesions. (See *Leprosy of Cornea*, page 515.) In most instances catarrhal conjunctivitis results from lagophthalmos and exposure due to cicatricial changes in the eyelids; but primary conjunctival involvement is usually due to infection from the neighboring skin or from nasal secretions.

Lepromas of the conjunctiva may appear either as isolated or

multiple miliary nodules. They are usually found on the bulbar conjunctiva near the limbus, are whitish in color, and tend to soften and ulcerate. Nodules at the limbus tend to spread into the cornea. King¹⁶⁵ described a form of nodular infiltration in the palpebral conjunctiva of the upper eyelid. The course of this disease in all its forms is slow, and marked tissue destruction ensues.

OCULOGLANDULAR TULAREMIA

The ocular manifestations of this severe systemic disease include fulminating conjunctivitis characterized by marked palpebral edema and small yellowish ulcers. The ulcers are numerous, discrete, and tend to become necrotic. They are more common on the lower than on the upper tarsal conjunctiva, the bulbar conjunctiva being rarely involved.

In Theobald's case,²⁹⁴ there were eight punched-out soft ulcers on the lower palpebral conjunctiva; among these, deep beneath the bluish conjunctiva, there were three or four small yellowish nodules. Painful swelling of the submaxillary, preauricular, and cervical glands was present. She believes that the histologic picture is much like that of an infectious granuloma.

Healing is possible without cicatrization, but chronic conjunctivitis is apt to persist for many months. This disease, in its oculoglandular manifestations, has the same appearance as the other conditions grouped under the heading of Parinaud's syndrome.

PARINAUD'S OCULOGLANDULAR SYNDROME *

This clinical condition occurs chiefly in the younger age groups; it is typified by conjunctival vegetations, thickening of the eyelids, tributary adenopathy, and a mild systemic reaction. Spontaneous healing is the rule (Plate X, figs. 5, 6).

* Duke-Elder,⁷⁰ in his discussion of Parinaud's syndrome, states, ". . . of recent years . . . more and more aetiological factors are being discovered in different cases, and it is now quite obvious that the term has been used indiscriminately to describe many different clinical entities. Parinaud's conjunctivitis is therefore not a definite disease with a specific pathology and bacteriology, but a syndrome of symptoms." Under the heading of infective granulomatous conjunctivitis he includes Parinaud's oculoglandular syndrome, tubercle of the conjunctiva, leprosy of the conjunctiva, syphilitic conjunctivitis, tularens conjunctivitis, necrotic infectious conjunctivitis, conjunctivitis pseudotubercle rodentium, sporotrichosis and agricultural conjunctivitis.

Renard and Nataf²⁴⁹ described the biomicroscopic findings. They distinguished the conjunctival lesions, situated either on the upper or lower tarsal conjunctiva, as large, swollen, follicle-like globules. These are whitish in color and may be spherical or ovoid, single or lobulated. In this respect they differ markedly from the smaller translucent follicles of trachoma. Vascularization is marked, especially around the periphery and over the surface of these structures. Between the follicle-like excrescences there may be voluminous vegetations, formed by a conglomeration of papillae. The papillae have the typical central vascular buds. Necrosis may occur in the center of these vegetations.

Small, clear, lymphatic cysts may be found on the bulbar conjunctiva near the fornices. These cysts tend to decrease in size as they approach the limbal areas where they become continuous with the dilated lymphatic channels of the limbal palisade zone.

A severe type of monocular keratoconjunctivitis with lymphadenitis has been reported⁵³ as due to infection by the virus of lymphogranuloma venereum. In this case there was extensive chemosis of the eyelids and conjunctiva associated with destruction of the cornea. The virus was identified by tissue culture.

MacNie²⁰⁸ reported nine cases of Parinaud's oculoglandular syndrome in patients known to be infected with lymphogranuloma virus. In four cases the conjunctival secretion or excised conjunctival tissue contained the specific inclusion bodies. Clinically, it is recognized that syphilis and lymphogranuloma venereum may exist simultaneously.

Hence, in cases of acute follicular conjunctivitis in syphilitic patients the oculoglandular syndrome of Parinaud may develop. In all such instances a thorough search for the lymphogranuloma virus, a Frei test, and proctoscopy are indicated to establish the diagnosis. We have seen a young man under treatment for syphilis who suddenly developed acute follicular conjunctivitis with marked swelling of the preauricular glands. The conjunctivitis did not respond to local treatment and after several weeks a diagnosis of lymphogranuloma venereum was finally established.

PEMPHIGUS OF THE CONJUNCTIVA (ESSENTIAL SHRINKAGE
OF THE CONJUNCTIVA)

Pemphigus vulgaris is a rare disease of the skin and mucous membranes characterized by the formation of bullae and cicatrization. The conjunctiva is often involved; this is apparently due to an extension from the skin lesions of the face. The disease may occur in either an acute or a chronic form. I have seen a case of conjunctival pemphigus in which no skin lesions or other mucous membrane affection could be found. The cicatrization results from invasion and proliferation of the connective tissue in the subepithelial layers. At the onset it resembles catarrhal conjunctivitis but later a thick ropy mucous secretion appears. Blebs, if present, are seen as circular red spots covered by necrotic epithelium. It is believed that pressure of the eyelid tends to prevent formation of conjunctival blebs, which were absent in 25 per cent of the reported cases.

James¹⁵⁹ described the early biomicroscopic appearance of conjunctival pemphigus. He noted that the first changes consisted of perivascular thickening resembling fine white striae. These striae act as lines of traction when the conjunctiva is moved and enmesh the superficial vascular network. This process later results in irregularity of the vessel caliber and finally in macroscopic scarring. James believes that the vesicle formation is due to edema which raises and vacuolizes the overlying epithelium. The epithelium itself is then exfoliated and replaced by fibrous tissue. The corneal involvement which starts at the limbus and is followed by vascularization consists of superficial punctate, grayish infiltrates in Bowman's zone. Infiltration and vascularization of the deeper layers of the cornea follow. Emissary capillaries may then make their way to the surface of the sclerosed cornea. In the earlier stages the cornea has the appearance of an onion skin, whereas later it resembles crumpled parchment.

The changes in the conjunctiva cause symblepharon, which, in the end, may lead to posterior symblepharon or to obliteration of

the fornices (complete ankyloblepharon (Plate XI, figs. 1, 2)). In the later stages of the disease, obliteration of the glands is followed by dryness, resulting in a picture typical of xerosis. At this stage biomicroscopic examination reveals a parchment-like appearance of the entire conjunctiva, which may be thrown in folds concentric to the cornea, with the presence of fine capillaries coming to the surface.

ERYTHEMA MULTIFORME (EXUDATIVA) (HÉBRA)

Because of its severity erythema multiforme may resemble acute pemphigus or hemorrhagic measles. It is characterized by an acute onset with widespread polymorphous skin eruption, associated with general symptoms (headaches, fever, pains in the joints). It may be marked by intense pseudomembranous inflammation of the mucous membranes, especially of the mouth, pharynx, and eyes. The ocular changes vary from slight involvement, such as catarrhal conjunctivitis, to complications severe enough to cause corneal scarring, loss of vision, and even total destruction of the eye. Dire ocular sequelae occur with the pseudomembranous form of keratoconjunctivitis. Bailey⁸ described three cases with severe ocular manifestations. In all his cases there was extreme swelling of the eyelids, making separation difficult; purulent discharge with pseudomembrane formation; and intense conjunctival congestion and chemosis. The palpebral conjunctiva showed papillary hypertrophy, was raw and granular, and exhibited a number of necrotic bleeding spots. In two cases corneal ulceration developed with serious sequelae.

ERYTHEMA NODOSUM

This is an acute eruption characterized by the formation of large tense swellings involving the whole skin thickness. It occurs in children or young adults, and usually affects the skin of the legs. The arms and face are less commonly involved. It is accompanied by symptoms resembling those of grippe, such as malaise, fever and pains in the joints. Several crops of eruptions may appear. However,

PLATE XI

FIG. 1. Pemphigus. Diffuse illumination. Folds and skinlike character of the bulbar conjunctiva, fornices obliterated, cornea densely scarred and vascularized.

FIG. 2. Biomicroscopic appearance of the lesions shown in Figure 1. Direct focal illumination at the limbus; beam is focused partly on the conjunctiva and partly on the cornea. 40 \times .

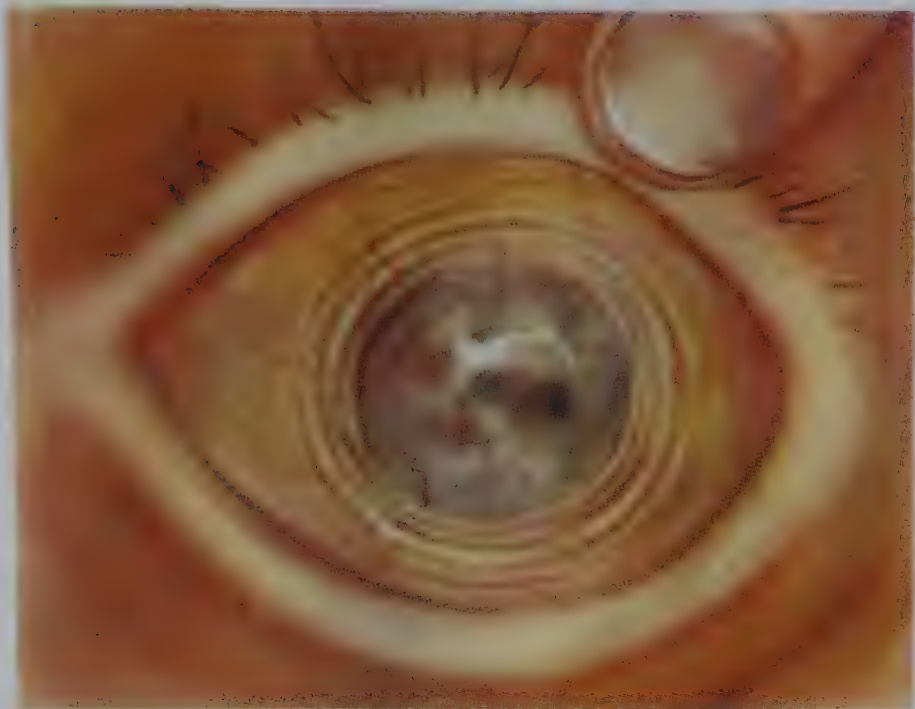
FIG. 3. Conjunctival and episcleral lesions in erythema nodosa. Two nodular swellings in the bulbar conjunctiva. Optic section reveals their deep situation in the conjunctiva and accompanying vascular congestion.

FIG. 4. Scar at the limbus following sulphuric acid burn. Note traction folds in the neighboring conjunctiva.

FIG. 5. Foreign bodies (coal dust) in the conjunctiva following an explosion.

FIG. 6. Chancre of the tarsal conjunctiva.

(Courtesy of Dr. Bruce Fralick.)



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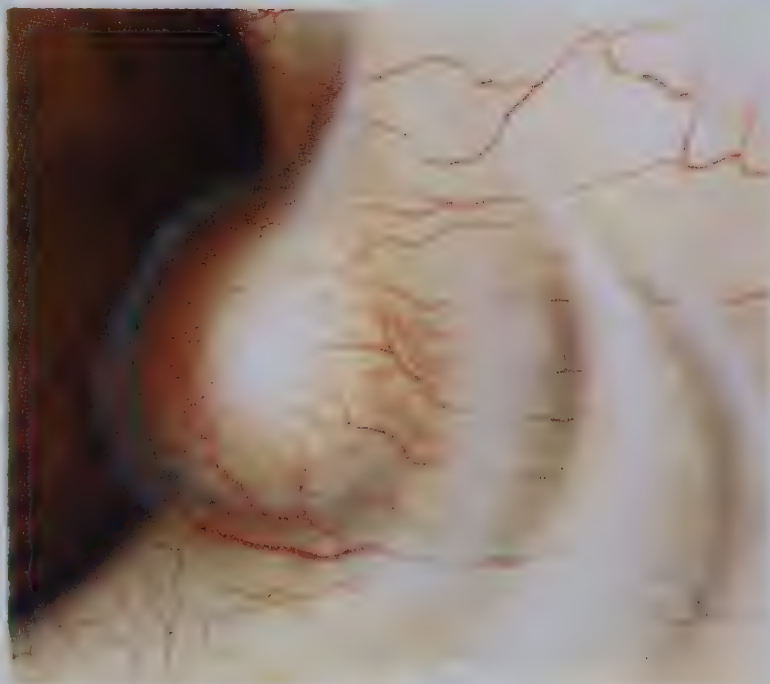
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all manifestations subside in a week or two with disappearance of the eruption. As a rule there are no visible sequelae and recurrences are rare.

The skin eruption of the eyelid regions may be accompanied by conjunctival involvement, which is characterized by the formation of vesicles or localized raised episcleral foci (Plate XI, fig. 3). In young children the edema may be so marked as to prevent opening of the eyelids. A considerable amount of secretion is present.

HYDROA VACCINIFORME

This disease is distinguished by a chronic recurrent vesicular eruption which has been shown to be related to exposure to actinic rays and consequent disturbance in porphyrin pigment metabolism. It appears to predominate in young males, in whom the exposed cutaneous areas are most affected. The ocular tissues involved are the eyelids, conjunctiva, cornea, and sclera. As pointed out by Stokes²⁸⁶ a great variety of lesions may occur. These include: (1) severe conjunctivitis with or without vesicles, either alone or in association with keratitis; (2) outbreaks of vesicles on the conjunctiva followed in some cases by ulcerating necrosis and formation of punched-out scleral scars; (3) a type resembling limbal vernal catarrh because of a peculiar hypertrophy of the perilimbal conjunctiva, and marked by pale, yellowish brown or grayish, raised, gelatinous efflorescences, but unaccompanied by the typical papillary hypertrophy (cobblestone granulations) of the tarsus.

Corneal complications result from the formation of vesicles which rupture, causing epithelial denudation, ulceration, and sclerosing keratitis; corneal opacities of all degrees may follow.

CONJUNCTIVITIS NODOSA

Conjunctival nodosa usually occurs during the summer months from the implantation of insect hairs or barbs, especially caterpillar hairs or plant hairs. These may excite a severe lesion not only of the conjunctiva and cornea but also of the intra-ocular tissues. Saemisch called the lesion "ophthalmia nodosa"; it had previously been known

as "pseudotuberculosis" because of the resemblance of the nodular lesions found in this disease to those of genuine tuberculosis. Many of the caterpillar hairs have fine barbs and spires directed toward the tip of the hair, which facilitate the migration of the hairs into deeper structures. In mild cases in which the hairs are confined to the conjunctiva without penetrating it, a foreign body reaction or a catarrhal conjunctivitis may be the only manifestation. When the hairs penetrate the tissues a severer reaction may result with the formation of numerous flattened nodules in the conjunctiva. These nodules are grayish or yellow in color and gelatinous in appearance. Usually several weeks or months elapse between the initial irritation and the appearance of nodules.

Many cases have been reported in the literature of caterpillar hairs penetrating the cornea and producing violent iridocyclitis followed by formation of nodules in the iris. Remissions and exacerbations are the rule but complete recoveries have been known. The violent reaction has been attributed to the toxic nature of the chitinous material composing the caterpillar hair. Knapp¹⁶⁸ recently described a case of bilateral ophthalmia nodosa occurring in a young woman who had been exposed to plant hairs (hairy moss). There was a moderate degree of conjunctival edema and hyperemia. The upper bulbar conjunctiva presented many small yellow subconjunctival nodules, extending from the fornices to within 3 mm. of the limbus. Histologic examination revealed the presence of foreign body tubercles.

ACNE ROSACEA

Conjunctivitis associated with acne rosacea of the face is usually accompanied by scaly blepharitis. This may be followed by corneal and episcleral involvement (page 476). The conjunctiva may show diffuse hyperemia or nodule formation. In the hyperemic type the bulbar conjunctiva is more or less edematous and congested, with papillary hypertrophy of the tarsal conjunctiva. The nodular type is rare. Small opalescent nodules, resembling phlyctenules, appear on the bulbar conjunctiva. Each nodule is supplied by a ter-

minal network of enveloping capillaries, derived from larger varicose vessels. The nodules may ulcerate but they usually resolve without leaving scars. When nodules appear in the conjunctiva near the limbus, they may simulate episcleritis periodica fugax, in that they are of a transient nature and tend to recur. One of the complications of rosacea conjunctivitis is the extension of the affection into the cornea, followed by development of keratitis. This is characterized by marginal vascularization and sharply defined grayish infiltration, which may extend into the deeper layers (page 477).

TRAUMA OF THE CONJUNCTIVA

The biomicroscopic picture following trauma of the conjunctiva depends on the nature and force of the injury and the resulting damage. Epithelialization usually takes place rapidly over denuded areas. Slight injuries or the presence of chemically inert foreign bodies, which do not lacerate the tissue, cause only mild localized transient hyperemia. Hemorrhage following rupture of the vessels is common. The hemorrhage may occur intraconjunctivally or subconjunctivally. The loose attachment of the conjunctiva to the episclera allows a drop or two of blood from a vessel to spread over a considerable area, in a manner similar to the spreading of a drop of fluid compressed between two cover glasses (Plates V, fig. 5; VI, fig. 1). In severe subconjunctival hemorrhage, a pouchlike separation of the conjunctiva may occur. There is a tendency for the blood to migrate toward the limbus, where the superficial limbal spur stands out in exaggerated form as a whitish band or arc owing to the contrast in color between the cornea and bright red area of subconjunctival hemorrhage. In the beginning the color is bright red but it gradually changes to orange and yellowish brown hues. Shortly after hemorrhages occur, manifestations of absorption appear. Clear whitish perivascular tracks form along the lymphatic sheaths; later, the larger, so-called clearing-up stripes invade the main hemorrhage. These stripes are irregular in shape, and may cross one another, forming a design. Usually within two weeks all bright red blood vanishes. A faint brown residue of blood pigment, com-

posed of small brown dots, remains. This may disappear entirely without visible residual stain.

An incised wound, involving the entire conjunctival thickness, usually leaves an ivory-like scar, as a result of which the conjunctiva may become adherent to the sclera. Contraction of a scar often causes glistening, radiating folds (traction lines) in the surrounding conjunctiva. When there is marked epithelial denudation of the conjunctiva, symblepharon is likely to occur. Such a process frequently follows a chemical burn.

If a wound has penetrated the sclera, uveal pigment is visible. If the edges of the conjunctiva are poorly coapted, for example, after an operation for squint, exuberant granulations with the formation of polypoid excrescences frequently develop. Wounds in the tarsus may heal with minimal visible scarring. Conjunctival scars are usually devoid of vessels. Alajmo states that superficial conjunctival wounds can be differentiated from small scleral perforations by the fact that in perforations a slight, localized chemosis (sometimes associated with deep ecchymosis) appears. By means of the narrow beam this point is clearly demonstrated.

Depending on the degree of damage, burns of the conjunctiva from chemical caustics may result in marked cicatricial deformations.* Symblepharon and ankyloblepharon may follow initial chemosis and coagulation necrosis (Plates XI, fig. 4; XII, fig. 1). The biomicroscope is of great value in searching for particles of glass, which are either free or embedded in the conjunctiva. If glass is present, oscillation of the beam over a suspected area reveals a characteristic glistening appearance.

Innumerable varieties of foreign bodies may become embedded in the conjunctiva (e.g., coal, silica, gunpowder particles) as a result of occupational exposure or accidents (Plate XI, fig. 5). Telangiectasia of the conjunctival vessels, associated with scarring, may follow radiation therapy.

* I have recently seen an instance in which a typical case of keratoconjunctivitis sicca developed following a mild acid burn of the conjunctiva.

Chapter Seven

TUMORS OF THE CONJUNCTIVA

PRIMARY tumors of the conjunctiva are rare. Some authors classify conjunctival tumors as either benign or malignant, but owing to the fact that clinical differentiation of tumors on this basis is difficult and that malignant transformation of a hitherto benign growth may take place, a pathogenetic or histo-anatomic classification appears to be more accurate. Mawas, on the basis of an extensive study of conjunctival neoplasms, stressed the importance of biomicroscopy in the differential diagnosis.²¹⁷ He classified the various forms of conjunctival tumors according to their origin, and his classification will be followed here.

EPITHELIAL TUMORS

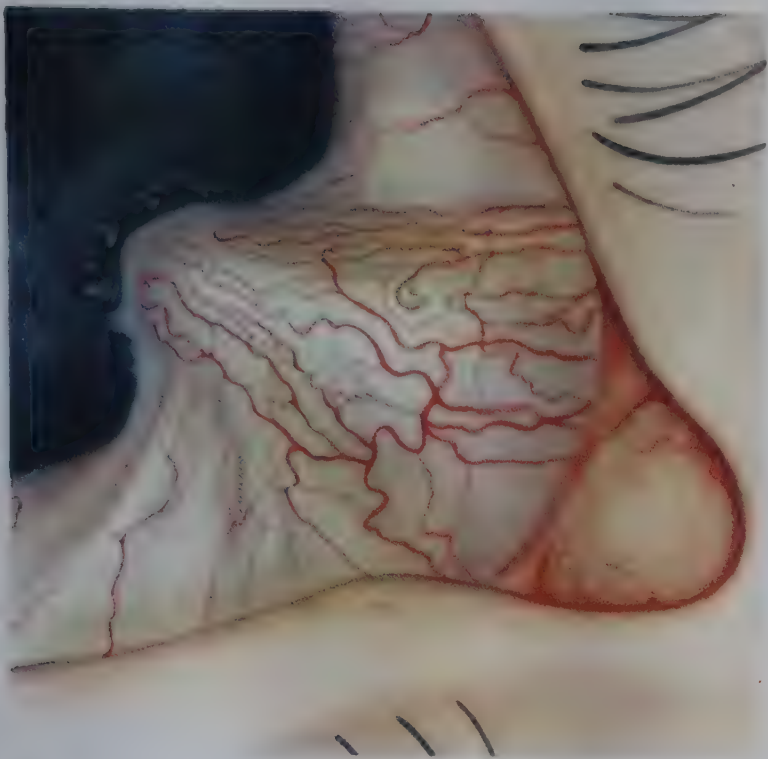
PAPILLOMA

Papilloma is most often situated on the palpebral conjunctiva, in the fornices, or on the caruncle, more rarely on the bulbar conjunctiva (Plate XII, fig. 2). When it develops on the bulb, it usually adheres to the limbus where it may be localized as a movable mass; or it may spread from the conjunctiva to the cornea either adhering to or actually invading the latter; or, again it may begin at the limbus, without conjunctival involvement, and extend over the cornea with its surface reddish and bosselated. Papillomas are soft in consistency, pinkish in color, pedunculated, raspberry-like, and have digital projections. In most cases their mobility differentiates them from malignant growths.

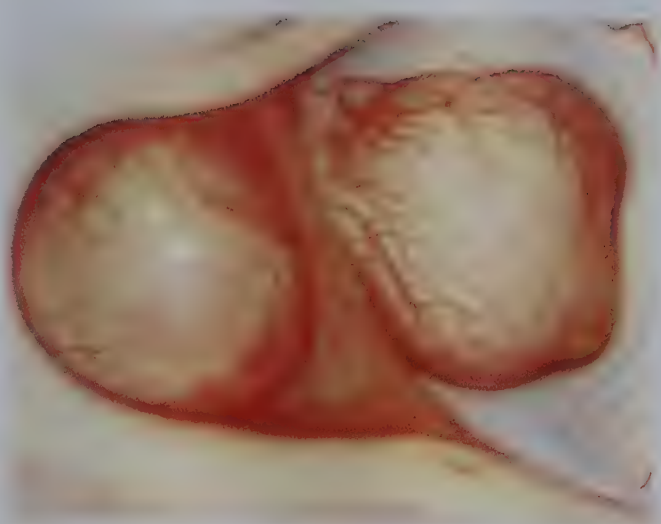
The biomicroscopic appearance of these growths is characterized by a regular, mosaic-like design of the summits of the papillomatous

PLATE XII

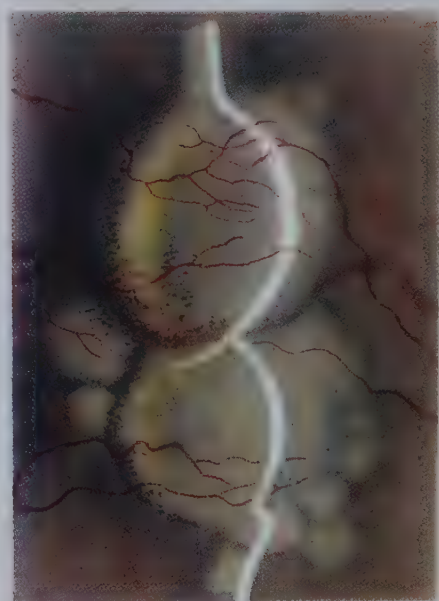
- FIG. 1. False pterygium following lye burn of the conjunctiva.
FIG. 2. Papilloma of the conjunctiva attached to plica semilunaris.
FIG. 3. Large conjunctival cyst in the lower fornix.
FIG. 4. Granular retention cysts of bulbar conjunctiva.
FIG. 5. Cystic melanoma of the caruncle.
FIG. 6. Optic section through filtrating cicatrix following trephine operation for glaucoma.



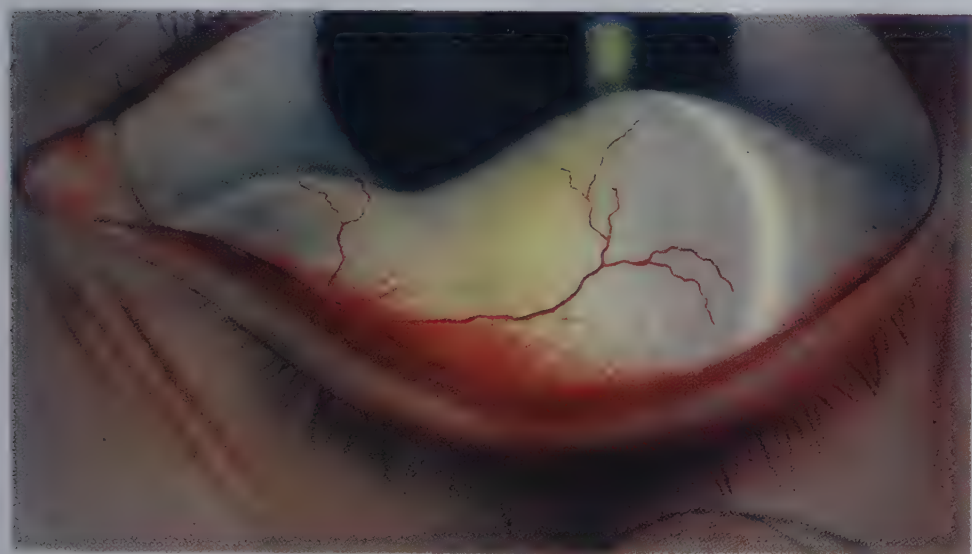
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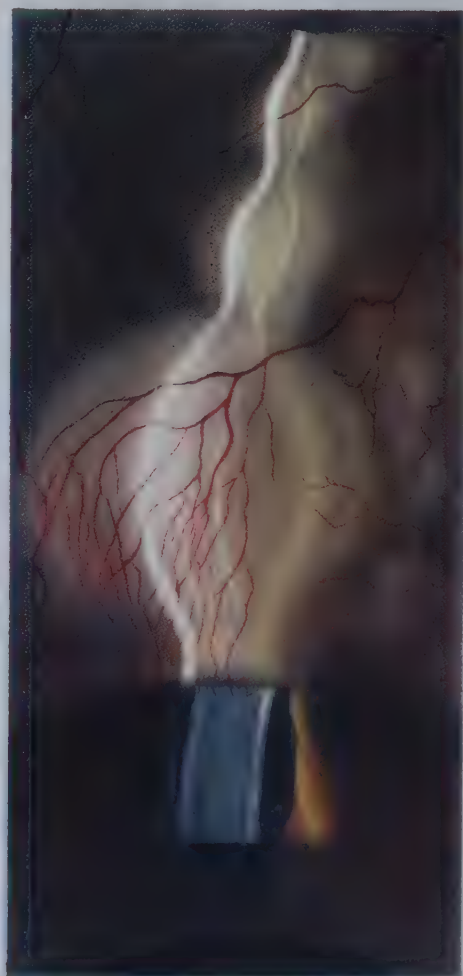


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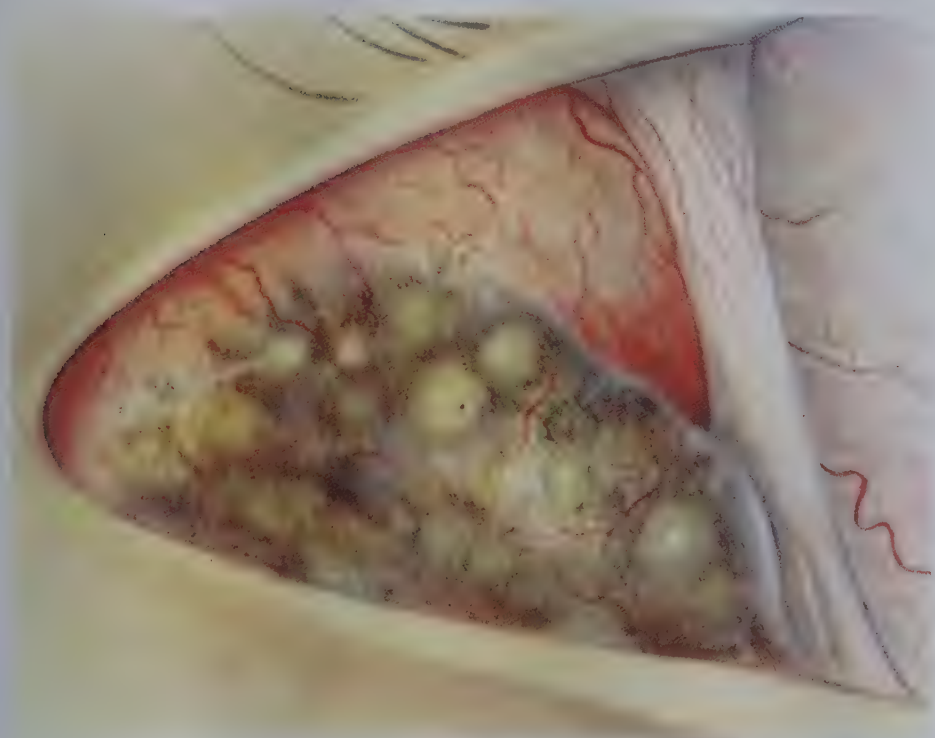


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excrescences, which may be quite large in size. The vascular supply is meager and has not the glomerular appearance of typical vascular formations of malignant epithelioma or granulation tissue.

CONJUNCTIVAL CYSTS

The more common conjunctival cysts are sequelae of trauma to the conjunctiva, for example, unrepaired lacerations, foreign bodies, operations for squint; or they may result from epithelial implantations, inversions, or adhesions of inflamed surfaces of conjunctival folds. Glandular retention cysts may develop from chronic inflammation, particularly in the tarsal conjunctiva and in the caruncle. A common site for retention cysts is in the lower retrotarsal folds. These cysts may attain fairly large dimensions, producing a prominence in the lower palpebral furrow. On eversion of the lower eyelid the cyst protrudes and is exposed to view (Plate XII, fig. 3). Branching blood vessels arborize over the surface. Retention cysts which form in the upper fornix generally arise from distention of Krause's glands.

With the narrow beam, cyst walls appear thin and glistening (Plate XII, fig. 4). Their contents are translucent. Chalky deposits are frequently found on the anterior surface. Archangelsky³ described a system of vessels with brushlike endings, surrounding filtrating cicatrices which developed following sclerocorneal trephination. The study of filtrating cicatrices following trephine operation (Elliot) presents interesting features. Biomicroscopic examination usually shows a conglomeration of small thin-walled cysts surrounded by scar tissue. Pigment granules may be found within the cysts (Plate XII, fig. 6) and frequently the scleral trephine opening is to be seen in the depth of the cysts. In some cases, downward dissection may cause a separation of the anterior corneal layers with resulting cystic changes. In others, adherence and traction of the conjunctiva may form a pseudopterygium. The absorptive effect of filtration may be seen upon instilling a drop of fluorescein; the color of the dye will disappear more rapidly around a filtrating cicatrix than on the rest of the globe (Seidel test).

Small cystlike dilatations, sometimes associated with pinguecula, occur in the interpalpebral zone of the exposed bulbar conjunctiva, surrounded by a delicate vascular network. Sometimes, associated with chronic conjunctival disease, multilocular or pedunculated lymphatic cysts, resembling lymphangiectasis, are found (Fig. 141). Rarely, cysts occur which are caused by parasitic infiltration, for example, *filaria loa* or *cysticercus*.

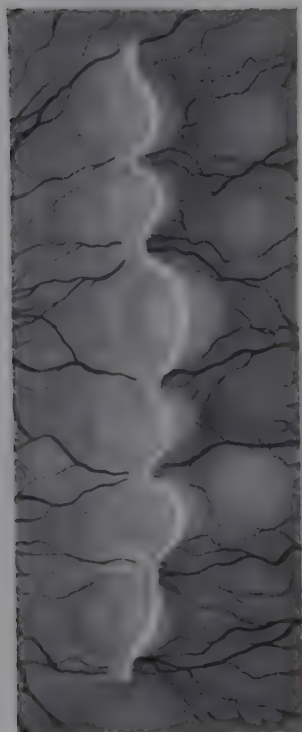


FIG. 141. Cysts of the bulbar conjunctiva in keratoconjunctivitis eczematosa.

EPITHELIOMA

Primary epithelioma of the conjunctiva is seldom seen. In most instances, it results from extension of a cutaneous epithelioma of the eyelid margin or of the caruncle. On occasion, its presence on the eyelid margin may be mistaken for an exuberant granulating chalazion.

However, the sclerocorneal limbus is a frequent site for these growths. When they appear at this site the designation of epibulbar carcinoma has been applied. The relative frequency of neoplastic lesions in this zone is attributed to the fact that a transition between the corneal and conjunctival epithelium exists at this point. The change in the basement membrane coupled with a physiologic metaplasia leads to a state of cellular "strain" which predisposes to neoplastic developments.

At the outset, an epithelioma at the limbus appears somewhat like a phlyctenule; it gradually increases in size, assuming a variety of forms — warty, berry-like, cauliflower-like, or sessile mushroomed masses (Figs. 142, 143). In all instances there is a firm immovable fixation to the underlying tissues.

The neoplasm tends to grow by superficial extension over the cornea and bulbar conjunctiva. Deeper invasion of these tissues occurs later. Because of predilection for the same site and an analogous type of superficial corneal infiltration, an epithelioma may simulate a pterygium.

Mawas has stressed the characteristic, almost pathognomonic, bio-microscopic appearance of the vascularization of epitheliomas. It differs from that of other neoplasms in that each epithelial lobule has

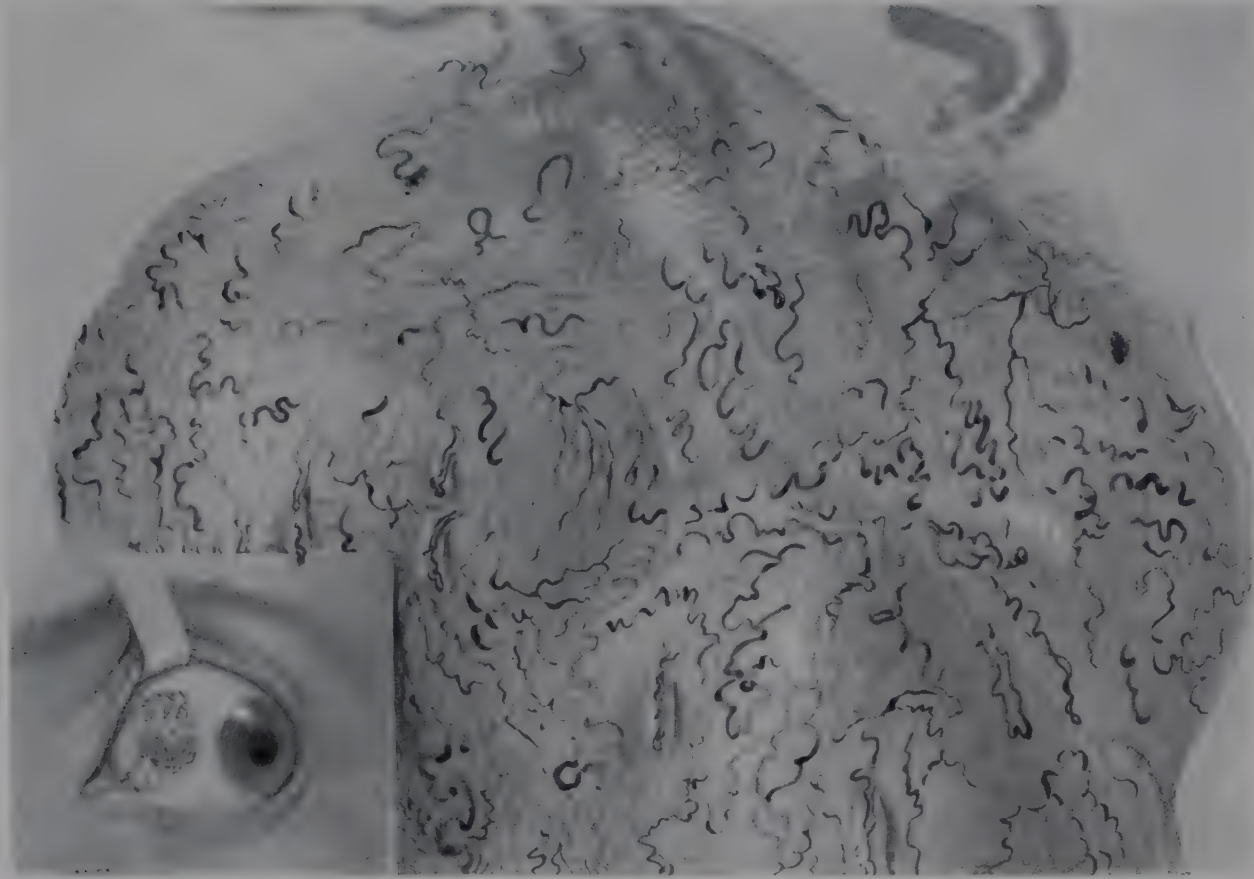


FIG. 142. Enlarged view of epithelioma of the bulbar conjunctiva, showing characteristic superficial vascularization. Insert shows macroscopic view and location of lesion. (Courtesy of Dr. A. B. Reese.)

a central vascular axis, formed by capillary trunks. The capillary entering the lobule mounts to the summit, curves upon itself and descends along the same route. In lobules, which are more developed, a glomerulus of capillary branches forms a budlike collection at the summit of each lobule. This vascular formation is typical of epitheliomas. The basal capillaries form a flat network which by anastomosis unites adjacent lobules. Mawas recommends vital staining with methylene blue in order to specifically stain nerve filaments and lymph channels. With the aid of ultraviolet light epithelial pearl formation may be revealed as fluorescent spots.

Intra-epithelial Epithelioma of Cornea and Conjunctiva (Bowen's Disease). McGavie^{203a} described five cases of intra-epithelial epithelioma of the cornea; although a rare condition, it must be differentiated from pannus, epithelial proliferation following radiation,

fatty degeneration of the cornea, xeroderma pigmentosum, and so forth (Fig. 144). Bowen originally reported two cases occurring in the skin, and considered it to be a precancerous dyskeratosis. In

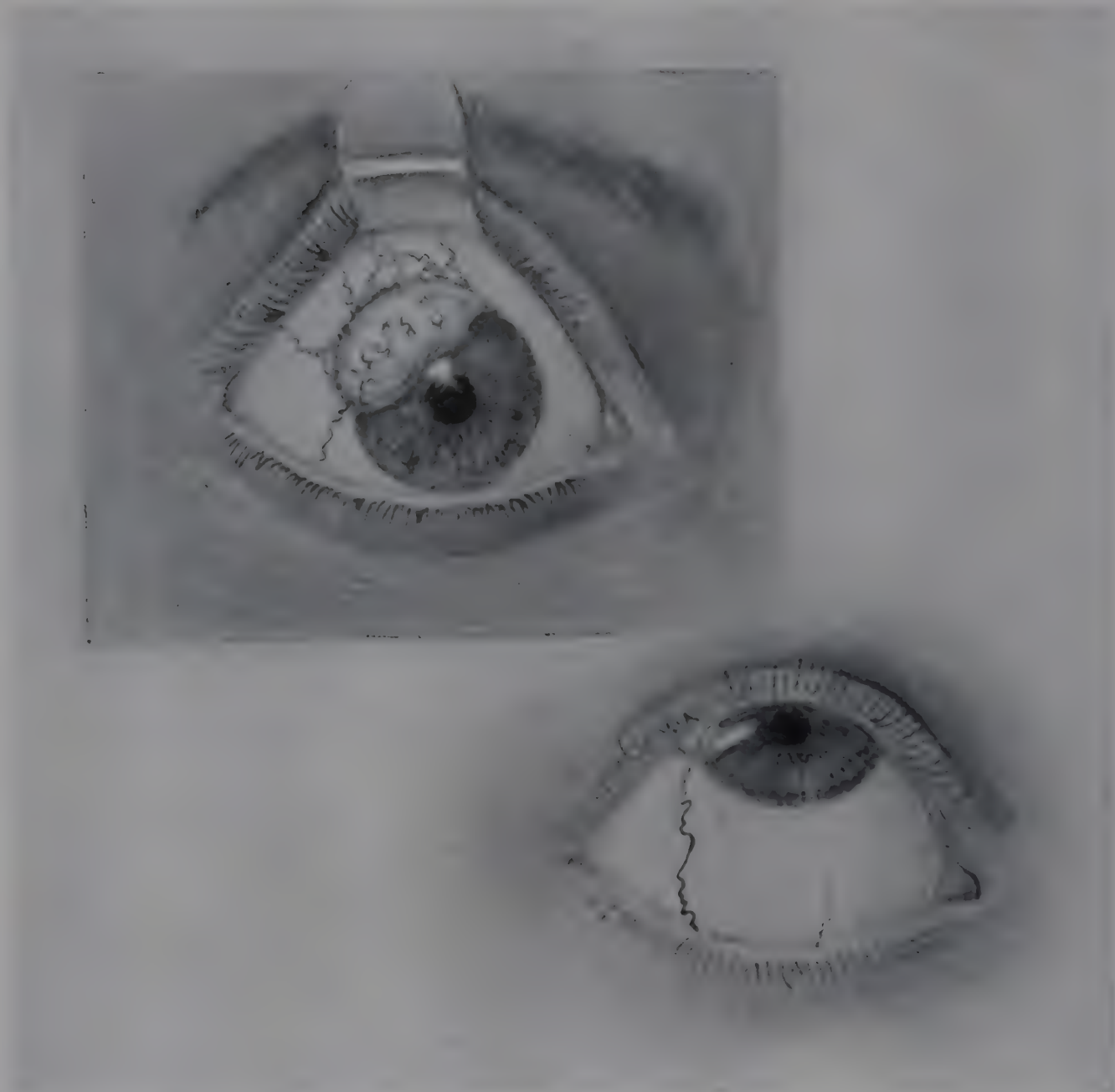


FIG. 143. Epithelioma of the limbus. (Courtesy of Dr. A. B. Reese.)

describing its clinical appearance McGavie states: "The lesions which we call Bowen's disease appear on the cornea and conjunctiva as slightly elevated, diffuse, sometimes multiple, highly vascularized patches of reddish gray, gelatinous tissue. In one patient whose bulbar and palpebral conjunctivas were extensively involved, the new growth had a yellowish gray membranous appearance. These tumors arise from epithelium and may remain entirely within the epithelium for years without breaking through the basement membrane to



FIG. 144. Intra-epithelioma of the conjunctiva (Bowen's disease). A. Case 1: Clinical appearance of pannus with intra-epithelial epithelioma covering the upper one-half of the cornea. B. Same as A with the patient looking down. C. Case 1: Appearance of the cornea after removal of the pannus and epithelial tumor. Bowman's membrane was not injured. D. Case 2: Clinical appearance of intra-epithelial tumor of the cornea. Note the vascularity of the lesion. (McGavie. *American Journal of Ophthalmology*, 25:167, 1942.)

show the usual tendency of epitheliomas to invade or metastasize. . . . In none of the cases was there evidence of invasion or metastasis." He concludes that:

"1. Bowen's disease is an intra-epithelial epithelioma which shows lateral spread in situ and is very slow to invade or metastasize. The histopathologic picture varies from ordinary epithelioma in situ to Bowenoid epithelioma.

2. The diagnosis depends on microscopic examination, but may be suspected clinically.

3. Complete surgical excision is the treatment of choice. Inadequate treatment by radiation, cautery, or chemical application is to be avoided.

4. Ocular involvement by Bowen's disease is here reported for the first time."

Through the courtesy of Dr. G. N. Wise and Dr. John H. Dunnington, I am including a case (Fig. 145) which will be reported on more extensively by Dr. Wise.

The patient was an Italian male, sixty-two years old, who had had recurrent attacks of inflammation of both eyes, periodically since the age of seventeen. These attacks have never been diagnosed. The present growth had been noted on the right eye three months prior to its removal. There had been no pain, the only complaints being the presence of the mass and the further decrease in vision. The tumor appeared to be removed in toto and the pathologic report showed it to be a Bowen intra-epithelial epithelioma of the cornea. Grossly, the lesion appeared to have three fleshy, pink, limbal nodules at 9, 12, and 3 o'clock, which were freely movable over the underlying limbus, and a diffuse irregular connected white tumor mass which covered most of the center of the cornea.

NEURO-ECTODERMAL TUMORS

The controversy concerning the origin and interrelationships of nevus, malignant melanoma, and melanosa sarcoma or leukosa sarcoma has not as yet been resolved. As pointed out by Duke-Elder,⁶⁹ "variations occur in structure, the significance of which is difficult

to assess while different types of cell formation may enter into the composition of one tumor. This has resulted in diversity of nomenclature and confused descriptions of similar neoplasms." Masson²¹²



FIG. 145. Intra-epithelioma of the conjunctiva (Bowen's disease). (Courtesy of Drs. G. N. Weiss and J. H. Dunnington.)

maintains that these tumors are neither ectodermal nor mesodermal but neuro-ectodermal and associated with the terminal neuro-apparatus. Theobald²⁹⁵ also considers that choroidal melanomas are of neural origin. As far back as 1882, von Recklinghausen pointed out a relationship between nevi and neurofibromatosis. In our discussion these tumors are included in one group — neuro-ectodermal.

NEVUS

This is one of the most common benign conjunctival tumors. It is considered to be of congenital neural origin, but may not make its appearance until some time after birth. The tumors usually grow slowly in childhood, becoming stationary in adolescence; or they may appear during pubescence, growing larger and then suddenly becoming quiescent. Although malignant transformation rarely occurs, its possibility must be kept in mind. According to Ewing,⁵⁸ such a transformation may take place as a consequence of irritation of simple traumatic, nervous, or more complex origin. The quiescent nevus cells may be incited to overgrowth and renewed pigment production, developing into a malignant localized tumor. The sclero-corneal limbus is the most common site of nevi. Frequent invasion of the cornea has been noted. Other sites, in which these tumors are found with some frequency, are the bulbar conjunctiva, especially in the exposed interpalpebral zone; the plica semilunaris; and the caruncle. The tarsal conjunctiva and the eyelid margins are less often affected. Cutaneous nevi, occurring coincidentally, are seen in many cases.

Conjunctival nevi may be nonpigmented or pigmented. The former variety is yellowish pink, translucent, and usually inconspicuous; but at times some yellowish brown pigment may be seen, which imparts a yellowish tinge to the growth (Plate XIII, figs. 5, 6). At the limbus, the nevus is usually firmly attached to the underlying episcleral tissues but peripheral to this zone it moves freely with the conjunctiva. The pigmented form appears as dark brown or blackish spots of variable shape and dimensions (Plate XIII, figs. 1, 8, 9). They are flat or only slightly raised and are sharply contrasted against the scleral background. With the narrow beam it is possible to localize nevi just within or beneath the epithelium. The pigmentation may be either discrete or conglomerate but it is usually powdery in type (Plate XIII, figs. 1, 2).

Another common characteristic of poorly pigmented nevi, which may be seen with the biomicroscope, are small round clear vacuoles

or cysts, frequently intercommunicating or separated by delicate septa; in optic section an area of these cysts resembles a slice of Swiss cheese. The more heavily pigmented nevi are solid in consistency and have a mottled surface. Most nevi are vascularized but it is important to note that, although vascular channels encircle the tumor, enter or traverse it, they give off *no vascular buds* (Plate XIII, figs. 3, 4, 9).

Careful biomicroscopic examination may make it possible to differentiate a benign nevus from the malignant form. In spite of the fact that malignant transformation of a hitherto benign nevus probably occurs rarely, it is important to keep accurate records. Changes in size or vascularization of a nevus must be looked on with suspicion. The sudden appearance of neovascularization (buds or glomerulus formation) is a suspicious sign. I have observed a case in which transient conjunctivitis excited local congestion of the conjunctival vessels; dilated vessels remained in the vicinity of the nevus weeks after the inflammation had subsided. Such a vascular response might have been misinterpreted as a sign of malignancy except for the fact that no neovascularization appeared in the nevus itself.

The transformation of a benign nevus into a malignant nevus is usually a slow process. However, instances have been reported in which a rapid malignant change has been noted. Dupuy-Dutemps has emphasized that the appearance of certain subjective symptoms (signs of inflammation) in a case of benign nevus is an important indication of the onset of a malignant change. These are itching, smarting and burning at the site of the lesion.

MALIGNANT NEVUS (MALIGNANT MELANOMA)

In Reese's opinion²⁴⁸ many more malignant melanomas of the conjunctiva arise spontaneously or from an acquired melanosis than from a nevus (Fig. 146). He believes that the rarer malignant melanoma, which arises from a nevus, grows as a localized tumor mass, while the tumor which develops from an "acquired" melanosis tends to be diffuse. He described six cases in which precancerous

PLATE XIII

FIG. 1. Pigmented nevus at limbus in diffuse illumination showing extension of pigmentation into the cornea.

FIG. 2. Optic section through the conjunctival portion of the lesion in case shown in Figure 1.

FIG. 3. Cystic nevus at the limbus associated with milky corneal zone adjacent to the lesion.

FIG. 4. Optic section through cystic nevus shown in Figure 3, illustrating the extensive multiple cyst formation.

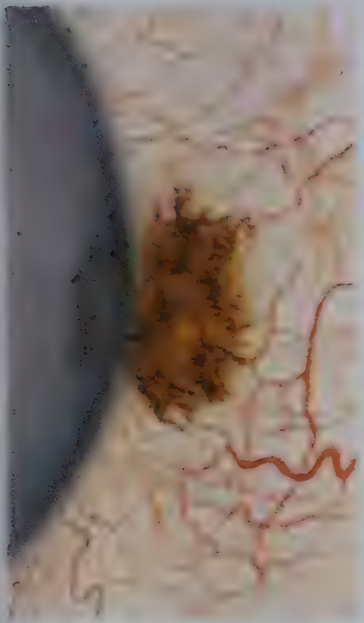
FIG. 5. Nonpigmented nevus at the limbus with typical vascularization (diffuse illumination).

FIG. 6. Optic section through nevus shown in Figure 5.

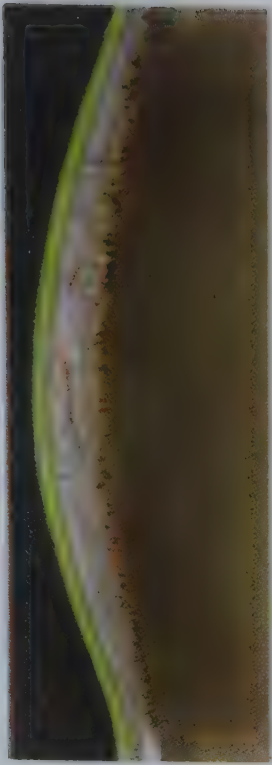
FIG. 7. Hemangioma of the limbus. (Courtesy of Dr. B. Rosenthal.)

FIG. 8. Pigmented nevus of the bulbar conjunctiva.

FIG. 9. Pigmented cystic melanoma. Large cyst in the center of the lesion.



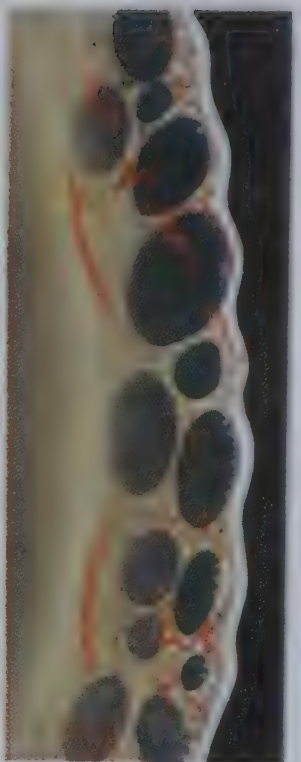
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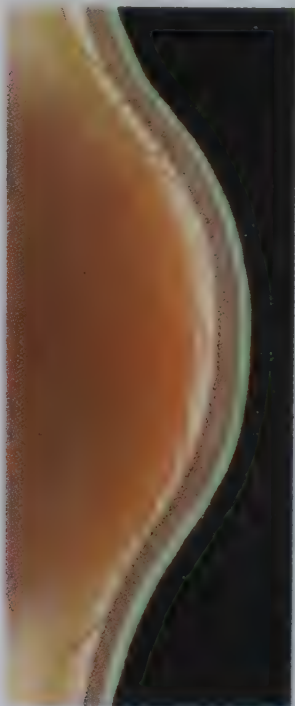
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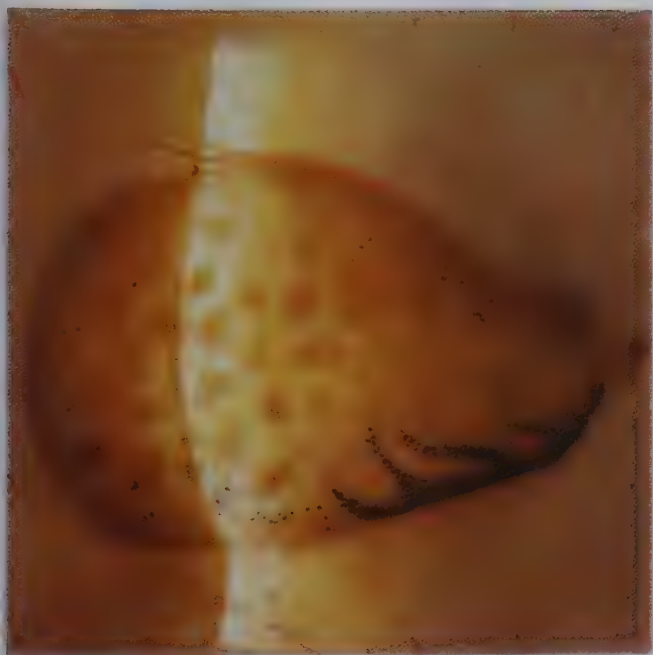
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melanosis of the conjunctiva was present for periods varying from seven to ten years before diffuse malignant changes were manifested. The malignant areas may remain flat, as in the precancerous stage.



FIG. 146. Malignant melanoma (conjunctiva).

Widespread flat involvement of the entire bulbar conjunctiva may occur without formation of a definite elevated mass. In such cases the tumor does not spread by direct continuity from one site only but rather from multiple scattered pre-existing foci. It should be noted that congenital melanosis of the conjunctiva and melanosis oculi, in which the pigmentation is confined to the sclera, must be differentiated from acquired precancerous melanosis.

Malignant melanoma appears as a conglomeration of small lobules; in the center of each lobule there is a tiny twisted capillary tuft branching from a larger vessel. There is a deeper, rich vascular network in the pigmented zones.

This pathognomonic vascularization, which simple nevi do not reveal, may be discerned with the biomicroscope. The optic section can demonstrate penetration of and attachment to the episclera.

When a limbal tumor invades the adjacent cornea, a milky white

zone of infiltration, visible in the superficial layers of the cornea, precedes the highly vascularized mass. This infiltration is similar to that occurring in pterygium. In certain instances malignant nevus must be differentiated from pterygium, pinguecula, phlycten, scleritis, or even embedded foreign bodies at the limbus. The neoplasm tends to spread over the conjunctiva fairly quickly, forming daughter-growths, especially on the palpebral conjunctiva.

Extension of malignant melanoma (melanosarcoma) of the uveal tract is a frequent occurrence. It is extremely important that early ophthalmoscopic and gonioscopic examination be made in order to find the original focus of the neoplasm. Extension of the tumor into the conjunctiva has been noted even after enucleation. The color of the tumor varies from a brownish to a deep black depending on the amount of pigmentation. These tumors usually have smooth surfaces and may occur in masses, which are more or less nodular, pedunculated, or sessile. At times, heavy black pigmentation precludes biomicroscopic examination of the depths of the mass or of the blood vessels.

Differentiation of nonpigmented melanomas from epitheliomas may be difficult. However, the latter are usually more exposed and lobulated and display a more or less characteristic vascularization (page 236). The sparsely pigmented tumors must be differentiated from intercalary staphyloma, granuloma, gumma, and tuberculoma; biopsy may be necessary before the diagnosis is established.

TUMORS OF VASCULAR OR LYMPHATIC ORIGIN (HEMOPOIETIC SYSTEM)

ANGIOMAS

These tumors are benign congenital growths. Two types are recognized: (1) hemangioma, which arises from the vascular system, and (2) lymphangioma, which derives from the lymphatics.

Hemangiomas. These appear as rounded, lobular masses, which vary in size, are movable, and of deep reddish or violet color. The capillary type consists of masses of dilated tortuous capillaries which

may occur in separate ill-defined groups, occasionally erectile (Fig. 147).

The tumor is most commonly found on the bulbar conjunctiva,

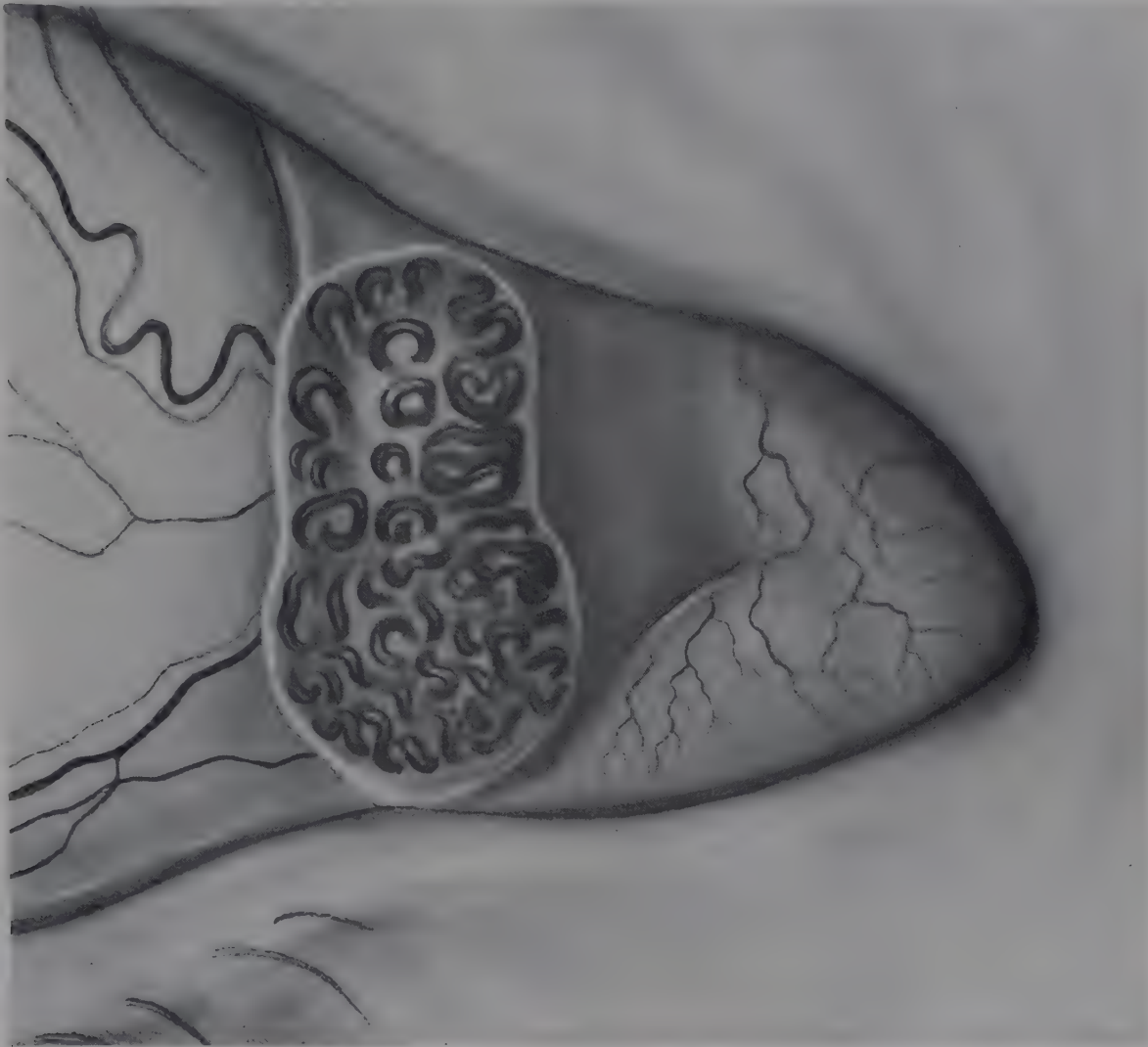


FIG. 147. Hemangioma of the caruncle.

frequently over the region of the recti muscles and in connection with the caruncle. I have seen hemangioma of the conjunctiva in a case associated with naevus flammeus of the face (Plate XXVI, fig. 5). Glaucoma developed in the involved eye. A distinct elevated mass, consisting of dilated and fusiform-shaped capillaries was situated on the temporal bulbar area. With optic section the deeper layers exhibited a yellowish, gelatine-like appearance. On the caruncle, hemangiomas appear either as small reddish masses formed by congeries of convoluted vessels or as solid yellowish pink tumors indistinguishable from unpigmented nevi. In a case of the latter type, seen by Dr. B. Rosenthal,²⁵⁵ the vascular nature of the tumor could be established only by biopsy (Plate XIII, fig. 7). For the most

part hemangiomas do not grow rapidly. Malignant transformation is rare. These tumors are markedly radiosensitive.

LYMPHANGIOMAS AND LYMPHANGIECTASES

These are lymphatic dilatations, found not uncommonly on the bulbar conjunctiva (Fig. 148). They appear as small cystlike ag-

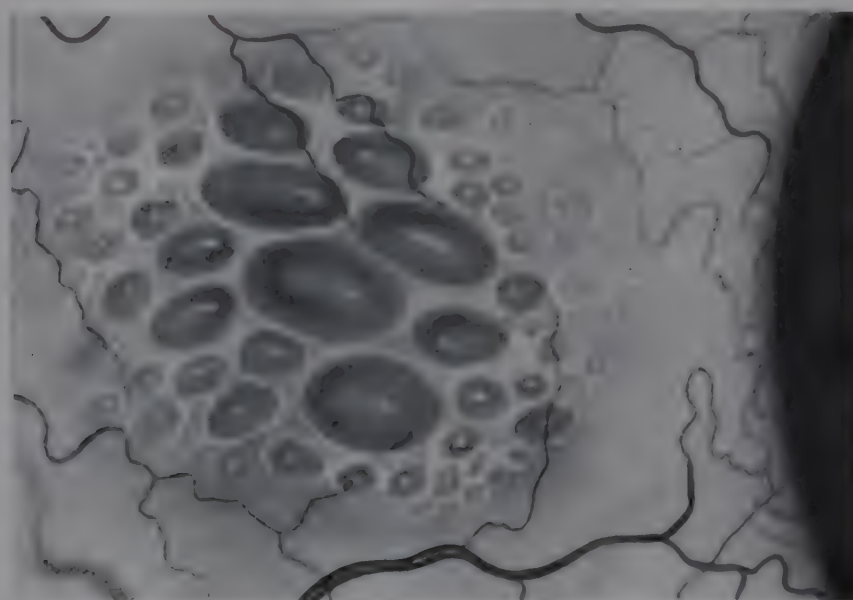


FIG. 148. Lymphangiectases of the bulbar conjunctiva.

glomerations, especially in the exposed interpalpebral conjunctival zones near the limbus, where the designation of lymphangiectasis is applied to them. Some form of irritation, such as friction of the eyelids, may be a factor in their causation, as well as a further stimulus to increase in size and number. The larger lymphatic varices, which are considered to be lymphangiomas, are rare.

Examination with optic section reveals the clear, water-like lymph contents and the absence of internal vascularization. The lesions are usually surrounded by a capillary network whose branches may extend over the surface. Pigment may be scattered over the surface, especially in the regions of the septal walls. Lymphangiomas may attain great size within the conjunctiva.

Lymphoma. Lymphoma of the conjunctiva may occur as part of a constitutional disturbance of the hemopoietic system, or it may appear entirely independent of any systemic disorder, manifesting itself as an infiltration of the palpebral and bulbar conjunctiva. At times, extension over the cornea from a limbal focus has been seen.

These tumors are firm, veal-like in consistency, and vary from a yellow to a pinkish color. With the biomicroscope they may be localized beneath the epithelium as yellowish masses with no definite structure discernible. Leinfelder and O'Brien,¹⁹² in a study of thirteen cases of ocular lymphoma associated with malignant lymphoid and hemopoietic diseases, found five cases of conjunctival lymphoma. In three cases the lesion appeared as a bulging discolored mass which protruded from the lower cul-de-sac on eversion of the eyelid. In two cases there were grayish red, fleshy nodules in the bulbar conjunctiva; in another case multiple large translucent follicles were found in the cul-de-sac and palpebral conjunctiva; in yet another, there was infiltration and enlargement of the caruncle and semilunar fold.

GRANULOMATOUS TUMORS (MESOBLASTIC)

Benign tumors composed of granulation tissue may occur in the conjunctiva either as simple protrusions (simple granuloma) or in polypoid forms. The former, which frequently develop in imperfectly joined conjunctival wounds (especially after muscle surgery) or following partial extrusion of a chalazion or a retained foreign body, appear as friable, highly vascularized granular masses, which are not epithelialized. Under the action of the eyelid the granulomatous mass may take on a polypoid shape, developing a pedicle. Biomicroscopy reveals a raspberry-like surface, each lobule consisting of small congeries of blood vessels which extend to the surface. This accounts for the ease with which these masses bleed. This vascular arrangement is not unlike that seen in malignant epitheliomas with which such masses may be confused, especially if they are large. Spontaneous expulsion or shriveling of most granulomatous masses is common, owing to the cicatricial contraction of surrounding tissues which pinches off the blood supply.

Polyps or fibromas of the conjunctiva occur as smooth-surfaced, pedunculated, soft or hard growths in the fornices, canthi, or on the tarsal conjunctivae. Although usually covered by epithelium, they may become ulcerated. The smoother, firmer polyps are poorly vascularized.

Chapter Eight

THE MARGINS OF THE EYELIDS

BIOMICROSCOPY OF THE EYELID MARGINS

THE eyelid margin is one of the many sites where transition of one form of epithelium into another occurs. That part of it which is nasal to the lacrimal punctum (bordering on the lacus lacrimalis) is smoothly rounded. The part which is temporal to the punctum and extends to the external canthus is flattened, thicker, and presents sharp anterior and posterior edges. Between these edges lies the flattened intermarginal zone. In order to study the margins slight eversion of the eyelids is necessary. In daylight, in the middle of this zone, a bluish gray pigmented, slightly depressed band (gray line) is visible. On the other hand, in direct focal illumination this color is changed because of the yellowish tint of the artificial illuminant and lowered diffraction. This change may be partially neutralized by the use of a bluish "daylight" filter. In daylight, with low magnification, the inner edge appears as a pale pinkish zone containing the line of orifices of the meibomian glands. In front of this is the bluish gray line (intermarginal sulcus) which serves as an important surgical landmark when splitting the eyelid. Anterior to this line is a pale linear zone just behind the cilia. The outer or ciliary zone contains the rows of cilia (Plate XIV, figs. 1, 2, 3, 4).

In Negroes, the pigmentation of the skin continues over the outer edge of the eyelid margin and extends to the inner edge of the gray line where the conjunctiva begins, forming a scalloped edge around the orifices of the meibomian glands. Similarly, a pigmented line occupying the outer border of the middle zone is frequently found in individuals of the so-called Latin races.

According to Panico, who also made a study of this subject from the viewpoint of biomicroscopy, it is advisable when examining the eyelids to moisten the skin with oil in order to make the epithelial

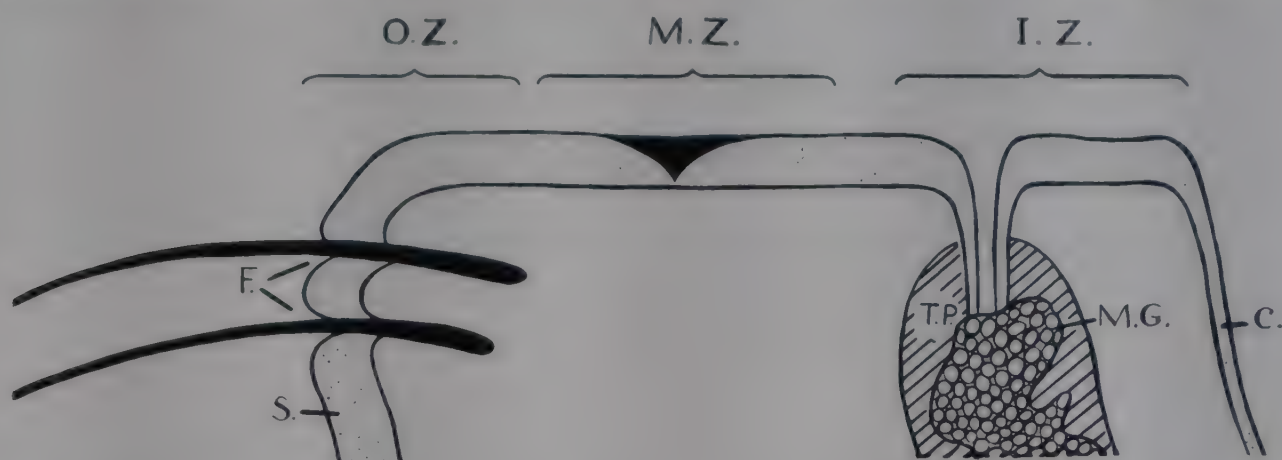


FIG. 149. Diagram of zone of eyelid margin. O.Z., Outer zone; M.Z., middle zone; I.Z., inner zone; C, conjunctiva; M.G., meibomian glands; S, skin; F, hair follicles; T.P., tarsal plate.

layer more transparent, and to use a yellow filter to increase the visibility of the marginal structures. The upper and lower marginal borders of the eyelids do not differ markedly in structure and, therefore, do not require individual description.

The eyelid margin may be divided into the following regions: (a) inner zone (conjunctival glandular region), situated between the posterior border of the eyelid margin and a line which passes in front of the openings of the meibomian glands; (b) middle zone (intermarginal sulcus of von Graefe), appearing as a fine bluish or brownish gray band and extending up to the zone occupied by the cilia; (c) outer zone or region of the cilia (Fig. 149).

Inner Zone. When the beam of light is directed on this region, the surface seems to be formed of a semitransparent tissue, resembling mother-of-pearl and having a pale pink tint owing to the presence of subjacent vessels. Some small red points may be observed; these points are small capillary loops advancing toward the surface, but still under the epithelium. Other small vessels are evident which derive from the conjunctival vessels and lie superficially. No more than one or two vessels are present in each interglandular space. With indirect illumination a number of flat grooves can be seen; Panico considered these to be rudimentary cutaneous papillae.

PLATE XIV

FIG. 1. Normal eyelid margin in young individual. O.Z., outer zone; M.Z., middle zone; I.Z., inner zone.

FIG. 2. Normal eyelid margin in older individual.

FIG. 3. Optic section of lower eyelid margin.

FIG. 4. Eyelid margin in the Negro. I.Z., inner zone; M.Z., middle zone; O.Z., outer zone.

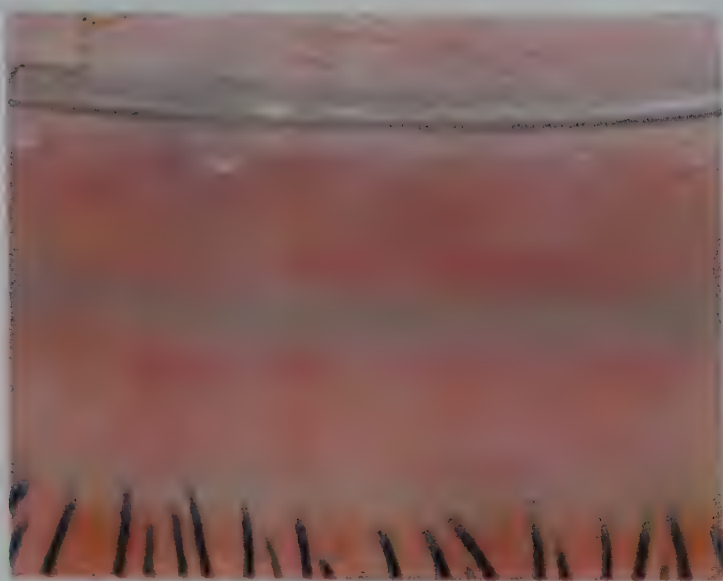
FIG. 5. Orifice of the meibomian gland. 60 X.

FIG. 6. Appearance of cilium in focal illumination. The transparency of the cortex seen in the middle portion of the shaft is due to reflection.

I.Z.

M.Z.

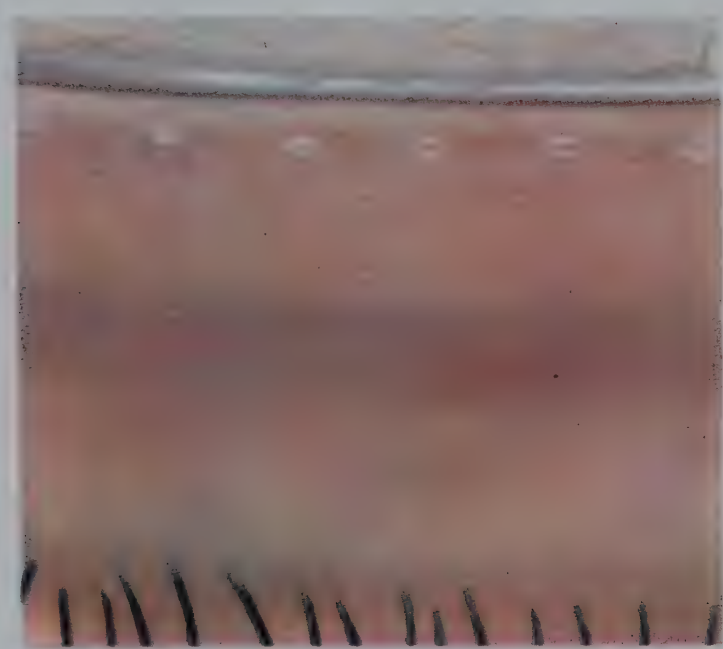
O.Z.



1



3



2

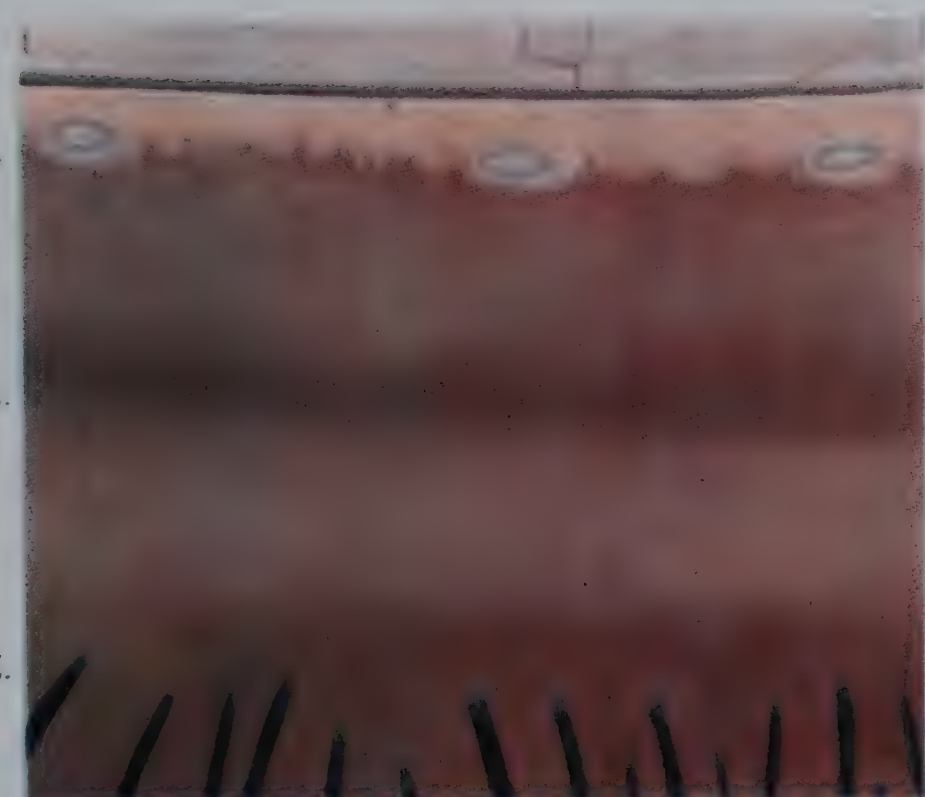


5

I.Z.

M.Z.

O.Z.



4



6

The outstanding feature of the outer region of this zone is the presence of the orifices of the meibomian glands, numbering thirty to forty in the upper eyelid and from twenty to thirty in the lower eyelid; they are surrounded by a white mother-of-pearl-like matrix, more transparent than the neighboring tissues (Plate XIV, fig. 5). These openings are usually closed or half closed, but on slight pressure they open, releasing a small drop of clear fluid. The openings are easily stained by instilling a drop of fluorescein in the conjunctival sac and allowing the excess to run over the eyelid margins. By Trantas' method of transillumination of the eyelid with Lange's lamp or a May light (diaphanoscopy) they appear as round luminous spots in a faintly glowing tissue. The rudimentary papillae, appearing in this zone, are more decisively outlined by transillumination. When stained with azur ii or fluorescein the dye accumulates in the orifices of the meibomian glands, accentuating their visibility and throwing them into sharp relief.

Middle Zone. This appears as a smooth and glistening, somewhat depressed band composed almost entirely of the bluish or brownish gray intermarginal line (gray line).

Outer Zone. In the outer zone the skin is more irregular, vessels are completely absent, except for an occasional large one in the deeper planes. The roots of the cilia are surrounded by a clear empty space seemingly filled with a semitransparent, gelatinous material, which may form a collar about the root of the cilium.

Dark eyelashes appear black in diffuse and proximal illumination (Plate XIV, fig. 6), but direct focal illumination reveals a yellowish central medullary cone, enveloped by a cortical sheath of a dark chestnut color. In direct focal illumination, red eyelashes are seen to be composed of a whitish medullary cone surrounded by a cortex, less transparent and of a golden hue.

Blond cilia largely resemble the red, the only difference being that the cortical sheath is of a light yellow color. In contrast to other hairs of the body the cilia are not as prone to turn gray with age. After injury or disease whitening of the cilia (canities) may be observed. White or gray cilia entirely lack pigmentation; the

medulla is opaque white and the cortical sheath transparent. Under high magnification they resemble icicles (Fig. 150).

There are about 150 cilia in the upper eyelid and they measure



FIG. 150. Difference between gray and black eyelashes under high magnification.

from 8 to 12 mm. in length; in the lower eyelid, the cilia are fewer and shorter, measuring only 6 to 8 mm. The cilia are cylindrical in shape in youth but become slightly flattened with senescence. The growth of a new cilium proceeds in the following way: As the old cilium approaches its senescence, a new, small and slender bud is

formed in the follicles. Slowly the diameter of the new cilium increases until it approaches that of the older. At the same time it also grows in length until it reaches two-thirds the length of the

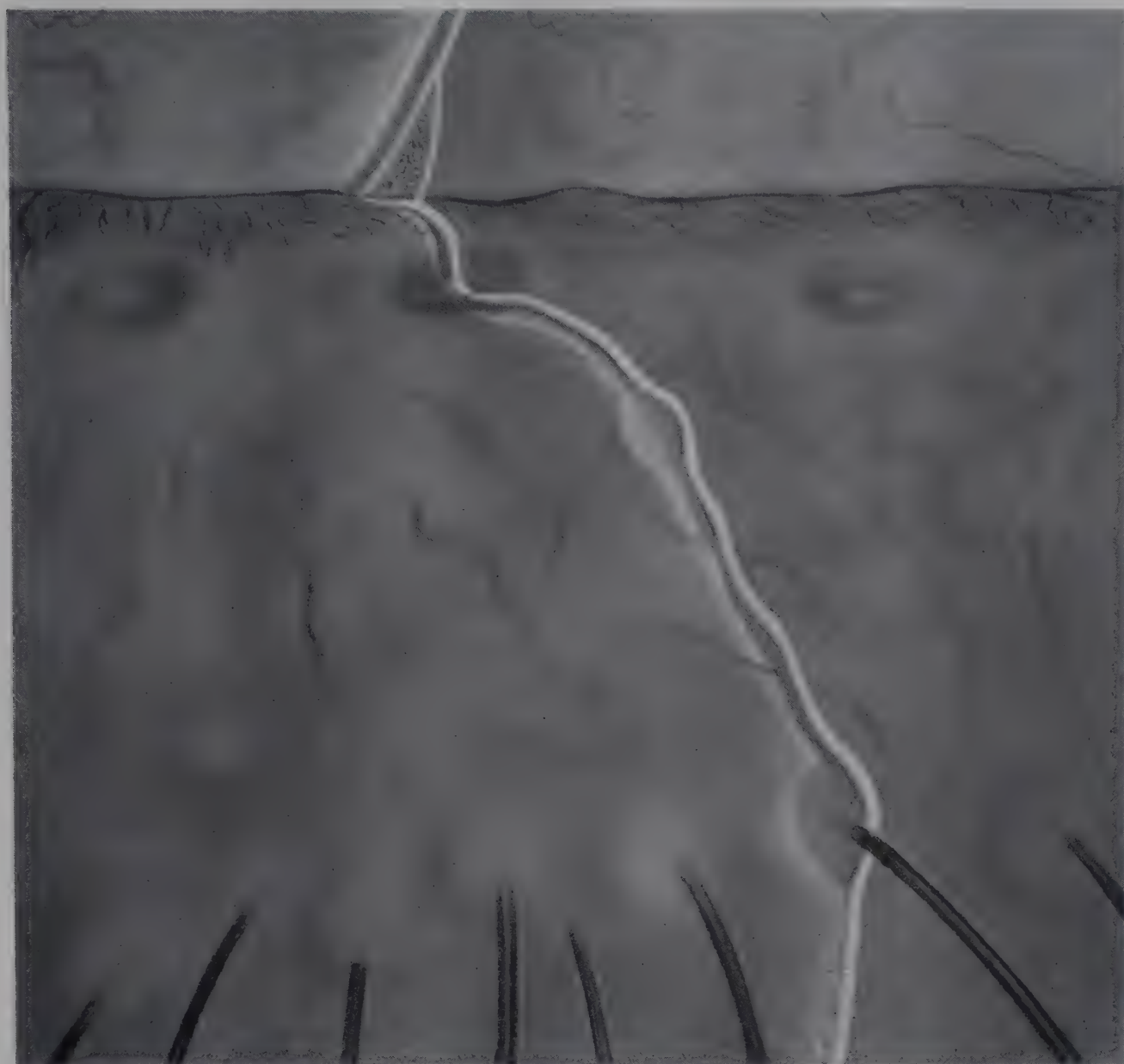


FIG. 151. Eyelid margin in senile individual showing vessels and cystic degeneration (direct focal and proximal illumination).

older cilium. Meanwhile the older cilium undergoes a regressive process, losing some of the pigment (turning from black to brown) and diminishing in size until it becomes loosened and is extruded by the newer cilium. The life cycle is from ninety to one hundred and fifty days.

In the aged a definite increase in the thickness of the eyelid margins occurs (Fig. 151). The vessels become more prominent and may be seen running across the zones just below the epithelium. The

semitransparent mother-of-pearl appearance is lost and is replaced by a denser yellowish color. A decrease in the number of cilia is also to be noted.

PATHOLOGIC ALTERATIONS OF THE MARGINS OF THE EYELID HYPEREMIA

Hyperemia of the eyelid border, which may lead to chronic blepharitis, is a common occurrence, particularly in individuals of light pigmentation (blond or reddish), who are hypersensitive to light or minor irritations. Acute allergic congestion of the eyelid margins and skin are known to follow hypersensitivity to certain foods, and drugs such as atropine. Biomicroscopic examination reveals capillary engorgement and suffusion of the superficial vessels with loss of the normal waxy appearance of the tissue.

In nevus flammeus of the face, the eyelid margin may be involved (Plate XV, fig. 2). Congeries of dilated and tortuous vessels are found below the surface.

CHRONIC BLEPHARITIS

Chronic blepharitis, a diffuse inflammation involving the whole eyelid border, may appear in a squamous (scaly) or ulcerative form. The former is characterized by the presence of small scales between and on the cilia (Plate XV, fig. 3). Occasionally, a yellowish crusting due to over-secretion of the glands may occur, with hyperemia but without ulceration of the skin. The cilia may be loosened and extruded. In the ulcerative variety, in addition to the hyperemia and crusting, actual superficial abscess formation of the hair follicles occurs. This is seen as a small pustular elevation surrounding the base of the cilium. As the condition progresses, the follicles may be largely destroyed, the cilia permanently lost, and their pits occupied by scar tissue (madarosis). A frequently seen intermediate stage is the presence of trichiasis owing to scar traction on the remaining follicles which causes a change in the direction of the cilia.

Chronic hypertrophic blepharitis with thickening of the eyelid

border may result in drooping or tylosis, especially of the upper eyelid. The lower eyelid may become everted (ectropion) and epiphora result from eversion of the punctum. This may further complicate the condition through excoriation of the skin with eczematous changes. At the inner edge of the eyelid margin, conjunctival papillary hypertrophy may be observed with the biomicroscope (Plate XV, fig. 4). Grossly, this appears as a red line. After marked eversion, this border loses its sharp outline and becomes rounded.

All forms of blepharitis are attended by marked increase in vascularization in which there is increase in the number of vessels running perpendicularly across the eyelid border just beneath the surface.

HORDEOLUM EXTERNUM

Hordeolum externum is a purulent infection of Zeis's glands (Plate XV, fig. 5). An acute pustular swelling of the margin occurs, marked by considerable inflammation. Spontaneous rupture at the eyelid margin commonly follows, a fact distinguishing external hordeolum from the internal variety, an infection of the meibomian glands, which usually ruptures through the tarsal conjunctiva. Differentiation from *chalazion* is easily made because the latter usually lies deep in the tarsus, is firm and round, not tender (unless secondarily infected), is of long duration and is rarely located at the eyelid margin. Eversion of the eyelid in most instances reveals a bluish gray or yellow area of thin conjunctiva over the chalazion, generally surrounded by increased conjunctival vascularization (Plate XV, fig. 6). In large chalazia optic section shows a thinned-out conjunctiva overlying a cystic zone.

PAPILLOMATOUS EXCRESCENCES

Papillomatous excrescences at the eyelid margin, a common development, may result from subacute infection of the marginal glands. These excrescences, which have the appearance of solid,

PLATE XV

FIG. 1. High power view of the punctum orifice of the lower eyelid.

FIG. 2. Nevus flammeus involving eyelid margin. Numerous dilated and tortuous vessels extending over the eyelid border.

FIG. 3. Chronic blepharitis. Lid margin is thickened with numerous vessels and cysts. Scales on the eyelashes.

FIG. 4. Chronic blepharitis showing papillary hypertrophy at the inner margin, scarring, cuticular thickening, and vascularization.

FIG. 5. Hordeolum externum.

FIG. 6. Conjunctival aspect of chalazion of the everted lower eyelid.



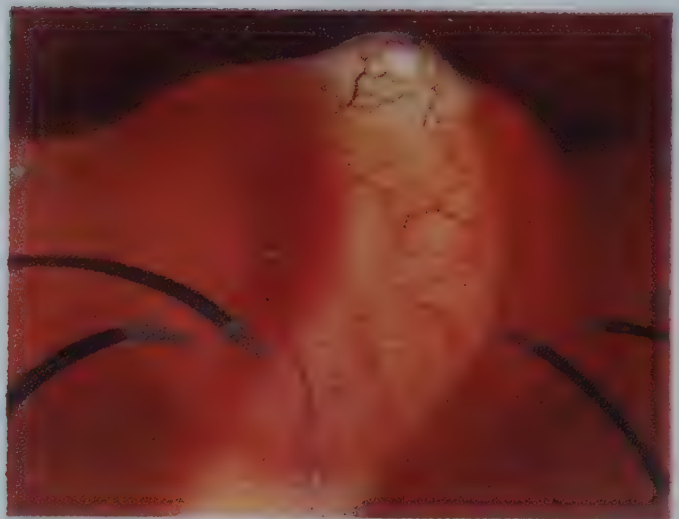
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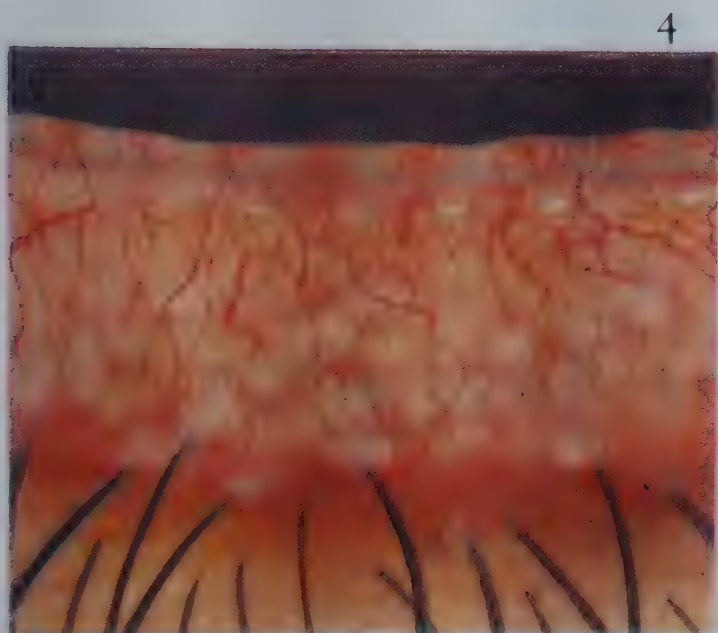


3



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6



4



rounded warty growths, often displace the adjacent cilia (Plate XVI, fig. 2). Rarely, *cornu cutaneum*, a large, elongated hornlike wart, may extend from the eyelid margin (Plate XVI, fig. 1).

Tumors of the eyelid margin include flat, *pigmented nevi*, or *nodular melanomas* occupying the intermarginal zone (Plate XVI, fig. 3). *Basal cell epithelioma* (rodent ulcer) may involve the contiguous eyelid margin, especially at the external canthus. This appears as an irregular superficial ulceration with a rough, crusted floor and firm, raised pearly border. The biomicroscope readily reveals the characteristic opalescent quality of the border as well as small friable bleeding points on the floor of the ulcer, which cause crusting. *Squamous cell carcinoma* is rare at the eyelid margin. When present it appears in the form of nodular or lobulated masses.

PARASITIC INFESTATION

Parasitic infestation of the margins of the eyelids may be caused by several species of parasitic organisms. The most common of these is the crab-louse, which is responsible for the condition known as *phthiriasis palpebrarum*. Owing to adhesion of the blackish nits of the crab-lice to the cilia, the outer eyelid border has a dark pigmented appearance. The biomicroscope discloses the translucent nits containing the black larvae, glued to the bases of the ciliae when the infestation is fresh or fastened along the shaft if it has been present for some time. Occasionally a free louse may be observed burrowing deeply into a hair follicle between the cilia. A rarer infestation³² is that with the hair-follicle mite, *acarus demodex folliculorum*, which is found within the hair follicles of the cilia and is thought to cause chronic blepharitis. There is a black deposit in the skin around the cilium as it emerges from the follicle. At times, a small transparent filament may be seen winding around the hair-shaft.

LACRIMAL PUNCTA

In order to see the lacrimal puncta, it is necessary to evert the eyelid. The puncta resemble craters. The lacrimal canaliculus lies

PLATE XVI

FIG. 1. Cornu cutaneum of the lower eyelid margin.

FIG. 2. Herpes zoster of the lower eyelid margin (outer canthus). A small papilloma is seen nasally.

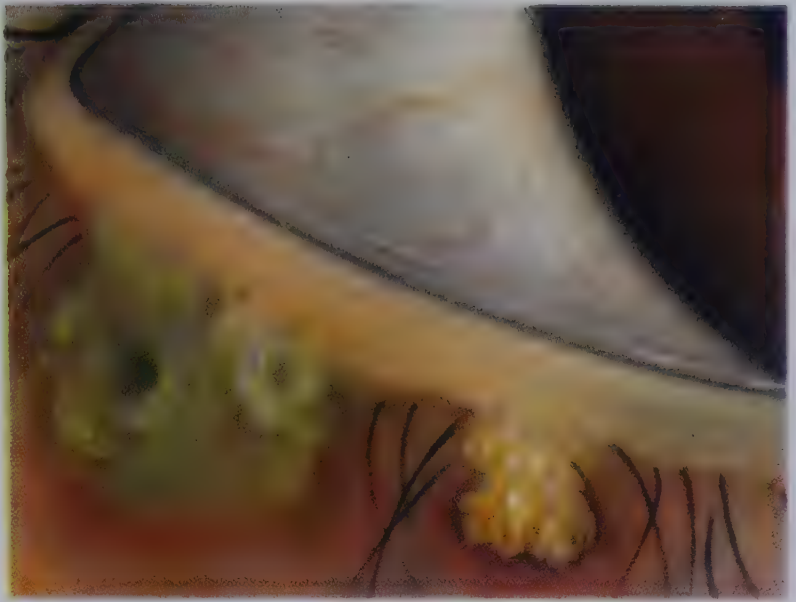
FIG. 3. Melanoma of the upper eyelid margin.

FIG. 4. Scleral plaque.

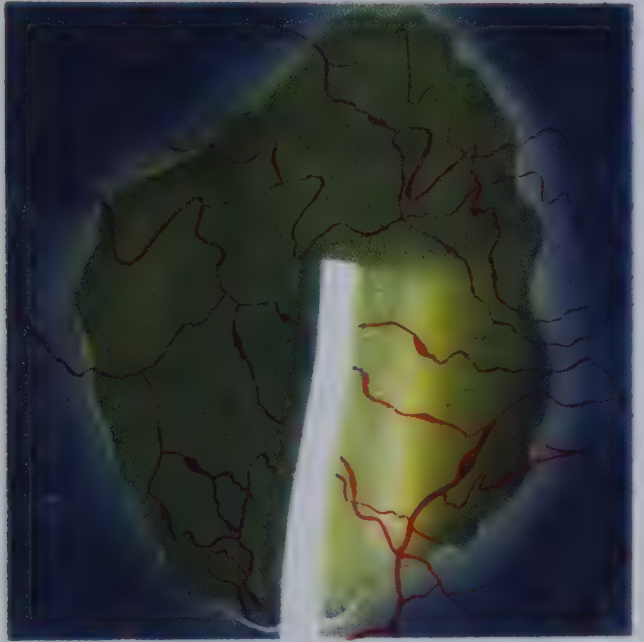
FIG. 5. Episcleritis. Optic section through conjunctiva. Note vascular congestion and separation of conjunctiva (dark area) from episclera.



1



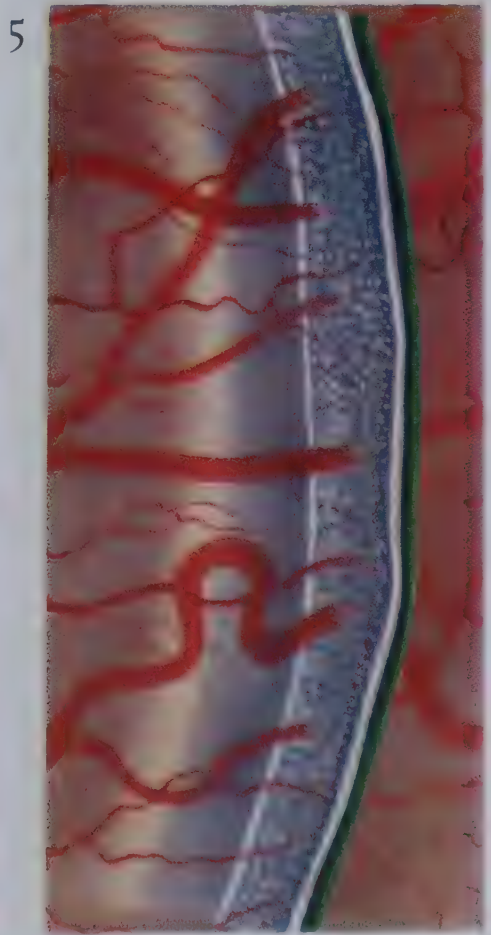
2



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3



5

in grayish white tissue which is completely avascular. The orifices of the canaliculi are surrounded by a ring of tissue of a pearly appearance, formed by intertwined elastic fibers. These fibers are so rigid that they keep the lacrimal puncta open. The shape of the orifice is ellipsoidal, with the major axis of the ellipse parallel to the margin of the eyelid (Plate XV, fig. 1). When vitally stained the orifices are filled with the dye and stand out as colored spots.

Inflammation of the canaliculi can be caused by bacterial or fungus (*streptothrix*) infections. The canaliculitis, which results, produces a pointing of the punctum. Pus or exudate mixed with mycelia is sometimes expelled spontaneously. Occasionally concretions of the canaliculus are formed and may be extruded by manipulation and pressure.

Not infrequently a loosened cilium becomes lodged in the punctum and acts as a source of irritation.

Chapter Nine

EPISCLERITIS AND SCLERITIS

EPISCLERITIS

LOCALIZED reaction in the episcleral tissue, commonly seen in rheumatic, syphilitic, tuberculous, or allergic conditions, frequently occurs in the exposed areas of the palpebral fissure. It is difficult to draw a sharp line between episcleritis and superficial scleritis. Episcleritis may begin as a flat form (e.g., episcleritis fugax) or as a local nodular focus. Lesions caused by herpes zoster ophthalmicus may produce a similar appearance. The condition is usually associated with pain. The nodular type may be confused with phlyctenular conjunctivitis, as both have a whitish or yellowish raised central area, resembling mother-of-pearl. Moreover, both are trellised and festooned by vascular arcades. However, a phlyctenule moves with the gliding conjunctiva, except at the limbus, and tends to ulcerate, whereas a nodule in the episclera is firmly attached to the underlying tissue and the overlying conjunctiva can be caused to move over it. In optic section, the raised episcleral nodule, which is composed of a yellowish substance, opaque to light, is covered by the conjunctiva.

In the flat form of episcleritis the conjunctiva over the involved area is usually raised (chemotic) so that a less relucant space is seen between it and the episclera (Plate XVI, fig. 5). The larger episcleral vessels appear faded owing to the screening action of the overlying raised, swollen and yellowish conjunctiva. The conjunctival vessels are engorged. At times, in chronic cases thinned out grayish depressions in the episclera and sclera covered by conjunctiva may be seen. These areas are generally oval, sharply outlined, and measure from 2 to 4 mm. in diameter. With optic section the depressed area has a gelatinoid appearance.

SCLERITIS

Clinically, scleritis presents few distinctive variations so that from the standpoint of the etiological diagnosis biomicroscopic examination is admittedly of secondary importance. Although nodules may develop they tend to be more extensive and diffuse than the nodule of episcleritis (Duke-Elder).

The lesion may be confined to a localized area or it may spread around the circumference of the globe (annular scleritis) or posteriorly. With the biomicroscope it will be seen that although the overlying conjunctival vessels are congested, marked engorgement of the episcleral vessels imparts a violaceous hue to the deeper and thickened tissues. Small, punctate dots (abscesses) are evident, which later tend to resolve. The condition runs a long chronic course with or without exacerbations. Seldom, if ever, does ulceration through the conjunctiva occur, and healing results in variable degrees of scleral thinning and scarring. Owing to the *vis a tergo* of the intraocular pressure, ectasia or even staphyloma of the thinned sclera may ensue. Optic section reveals a dark cicatrix owing to the fact that thinned and less relucant scarred sclera allows the underlying uvea to be visible. Scleritis, in contrast to episcleritis, may be accompanied by severe iridocyclitis, especially when the scleral lesion is near the limbus. In this case, corneal involvement is likely to occur, resulting in a sclerosing, keratitis (sclerokeratitis). (See leprous and tuberculous keratitis.)

A more severe and intractable form of scleritis, known as brawny scleritis, is characterized by diffuse, annular, gelatinous thickening, which causes the entire, visible part of the sclera to resemble cut liver. Histologically, an extensive lymphocytic infiltration is formed, which in some cases simulates the picture of syphilis, and in others, that of tuberculosis. Although most cases have been reported in the aged and in both eyes, I have seen typical brawny scleritis develop in one eye after repeated subconjunctival injections of oxycyanide solutions for the treatment of vitreous opacities. The thickened reddish scleral tissue pitted on pressure. A low grade cyclitis with

marked increase of intra-ocular tension necessitated removal of the eye in order to relieve pain. In this case the swelling encircling the cornea began at the limbus and extended as far back as could be inspected. The optic section revealed a congested and edematous conjunctiva overlying the thickened, fleshy, and nontransparent subconjunctival tissue which had a deep yellowish red hue.

SCLEROMALACIA PERFORANS

Rochat²⁵⁰ and van der Hoeve³¹⁵ described an apparently non-inflammatory condition of the sclera which may or may not be associated with chronic rheumatic arthritis and which is characterized by "the appearance of holes in the sclera, which can coalesce so that the sclera shows large gaps in which the uvea lies either covered by conjunctiva or bare." The condition was originally called *scleritis necroticans* by Rochat, but owing to the absence of inflammatory signs, van der Hoeve considered it a degenerative lesion and suggested the name, *scleromalacia perforans*. In their cases, the holes were found anywhere from the limbus to the equator, and it was remarkable that in most of them the uvea did not herniate.

SCLERAL PLAQUES

Scleral plaques is another interesting scleral defect described by Graves¹³³ and Culler.⁵² It usually consists of a dark, oval, sharply defined area covered by conjunctiva, and is situated 4 to 6 mm. from the limbus. Its long diameter measures from 3 to 5 mm. and the short diameter from 2 to 3 mm. Originally Graves referred to the defect as "bilateral (mesial) deficiency of the sclera," but later, (1940) he suggested that the word "mesial" be dropped since the condition can occur temporally as well. I have recently seen a case in which the condition was found in one eye only and on the temporal side. This defect occurs in older individuals and must not be confused with the smaller dark areas (holes?) in the sclera which permit passage of a perforating artery or an aberrant nerve. With the narrow beam it may be seen that the edges of such

defects are refractile, that they are subconjunctival, and that the defects themselves consist of a homogeneous hyaline-like substance having a yellowish tinge. Apparently the normal scleral substance is replaced by this material. In Plate XVI, fig. 4, it will be seen that there is little if any actual depression over the surface of the plaque.

Chapter Ten

THE NORMAL CORNEA; BIOMICROSCOPIC APPEARANCE

IN diffuse illumination, the cornea appears transparent to the unaided eye, but optically its surface is revealed by reflexes due to irregular reflection. Passage of a beam of light (the prefocal or postfocal portion) through the cornea results in the formation of a diffuse, grayish, slightly opalescent area where the beam cuts the cornea. However, when the exact focal part of the biomicroscopic light beam is employed a grayish, opalescent sharp-edged figure is formed, its shape corresponding to that of the beam. This phenomenon is caused by the irregular dispersion, scattering, and reflection of incident light by the internal structure of the laminated cornea. The more homogeneous (isotropic) a tissue is, the less the dispersion or scattering of light occurs, and the less marked is its opalescence as the focal light passes through. The cornea is an example of a tissue whose complex internal structure optically results in multitudinous variations of indices of refraction. This produces considerable dispersion of light. The cornea, therefore, must be considered as an optically heterogeneous (anisotropic) tissue. The property of dispersion and scattering of the light beam by the ocular media has been termed *relucency* (Graves).

Further investigation has shown that the amount of light which is dispersed and scattered by the transparent ocular tissues, and consequently the degree of opalescence or relucency, depends on the size of the component colloidal particles and on the wavelength of the transmitted light.

Histologically, the cornea consists of five demonstrable layers (Fig. 152). The narrow beam of the biomicroscope shows an ad-

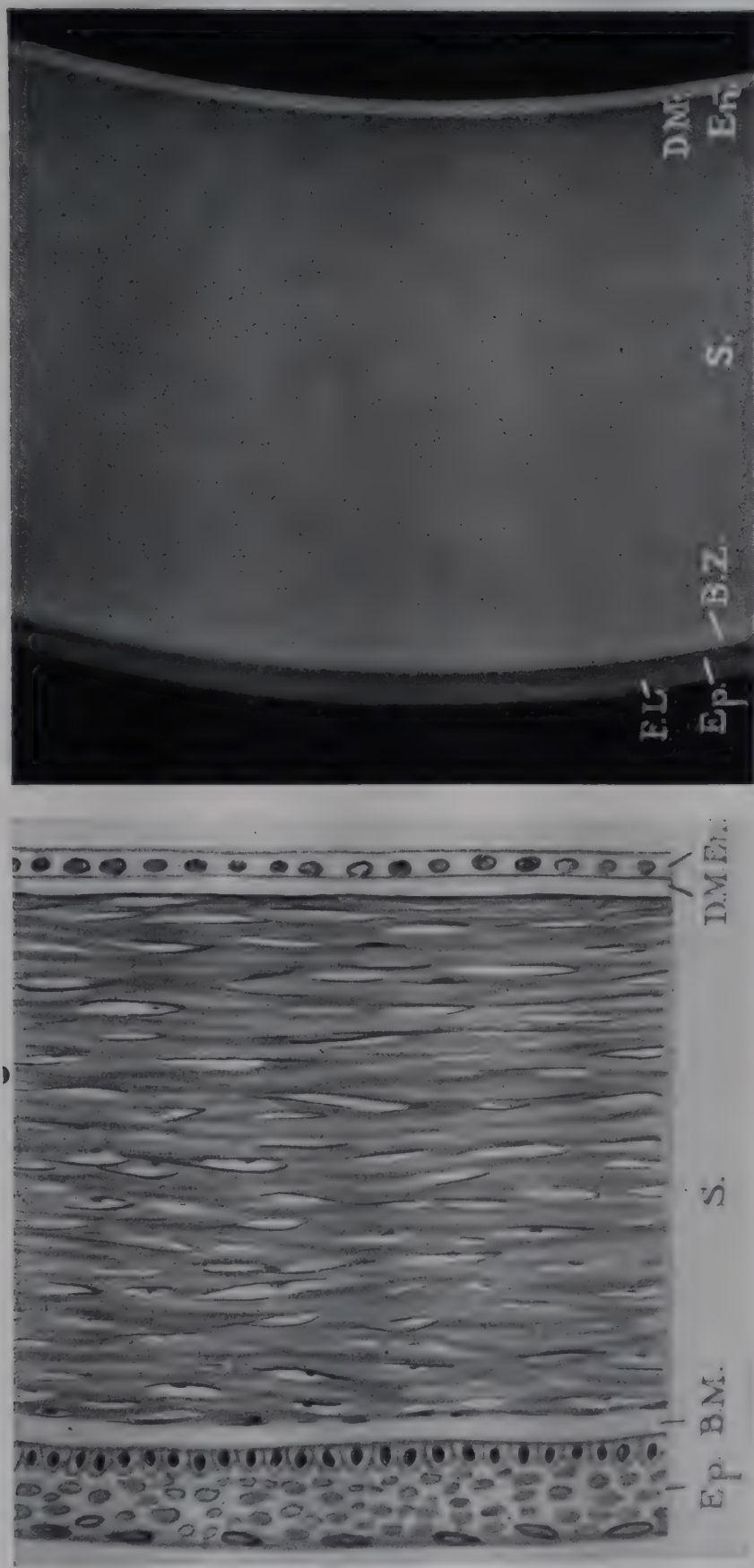


FIG. 152. A comparison between histologic (A) and optic (B) section of the cornea. *EL*, Film line; *Ep*, epithelium; *Bm*, Bowman's membrane; *Bz*, Bowman's zone; *S*, stroma; *Dm*, Descemet's membrane; *En*, endothelium.

ditional fluid layer in front of the epithelium, the so-called *pre-corneal film*. The cornea is an avascular tissue, except for the limbal regions. There are no true endothelial-lined lymphatic channels between the corneal lamellae — only potential spaces which may permit accumulation of lymph. The avascular cornea is richly supplied with nerve plexuses.

Using the various methods of illumination afforded by the biomicroscope, the question arises as to which of the normal corneal structures can be seen.*

THE STROMA

The corneal parallelepiped (D. F. I.) has a uniform bluish gray opalescence (Fig. 67), in which practically no internal design can be discerned, except in the region of the zones of specular reflection (Figs. 91, 96). As these areas are approached, an *internal specularity* (Graves) results; this produces small, glistening vertically striaform, irregular lines. These probably are the summation of reflexes from the individual lamellar surfaces, which act as mirrors.

Most authorities, in describing the structure of the corneal stroma as having a granite-like or marble-like design when seen in focal illumination, attribute this appearance to the presence of small irregular whitish lines. Koeppe described a corpuscular-like appearance which he considered as possibly due to a system of lacunae and lamellae.

Koby¹⁷⁵ states that "this marbling is due to the presence of small areas whiter than the rest, which have been called arachnoid corpuscles and which are possibly identical with the fixed cells of the cornea. Curiously enough the lamellar structure seen in histologic preparations does not appear in the examination of the living eye. It is probably an artifact caused by the reagents." The marble-like appearance in the corneal parallelepiped is not due to the visualiza-

* Experimental methods, e.g., the use of polarized, ultraviolet or monochromatic forms of illumination will not be included here. For the study of normal structures, direct focal illumination and specular reflection alone are of value. An exception to this is the use of retro-illumination and proximal illumination for studying the limbus.

tion of actual morphologic structures, but rather to an optical phenomenon (internal specularity). Fischer⁸⁵ pointed out that the ultramicroscope reveals the presence of longitudinal micellae in the corneal gel. However, there is no possibility of visualizing such submicroscopic structures with the biomicroscope.

CORNEAL NERVES

The corneal nerves are visible between the anterior and posterior faces of the block as short threadlike white lines (Figs. 153, 154). Typically, the nerves of the cornea are naked (nonmyelinated) axis cylinders, except at the limbus where the acquisition of a myelin sheath causes a tapering spearlike appearance; the nerves follow a course through the anterior layers of the cornea and tend to branch dichotomously. They are best localized in optic section with a wide angle of incidence. When the beam of light is thrown out of focus and the prefocal or postfocal portion of the beam is employed, the corneal nerves can be clearly seen as fine whitish threads, the courses of which can be traced for a considerable distance (Plate XVII, fig. 1). When examined with afocal illumination, the corneal nerves seem to be in a single plane in the anterior third of the cornea. In the region of the limbus, by indirect illumination, nerve fibers resembling spearlike projections enter the cornea in a radial direction. In the cornea proper, at the point of bifurcation of the fibers, small weblike or nodular corpuscles can be seen. Koeppe has interpreted these nodules as congenital neurofibromas. Trichotomous branching or T-shaped formation occurs. The number of corneal nerves has been variously estimated as between thirty and fifty.



FIG. 153. Corneal nerves in parallelepiped.

THE ANTERIOR SURFACE

The anterior surface of the parallelepiped (broad beam) corresponds to the anterior face of the cornea. In the normal cornea,

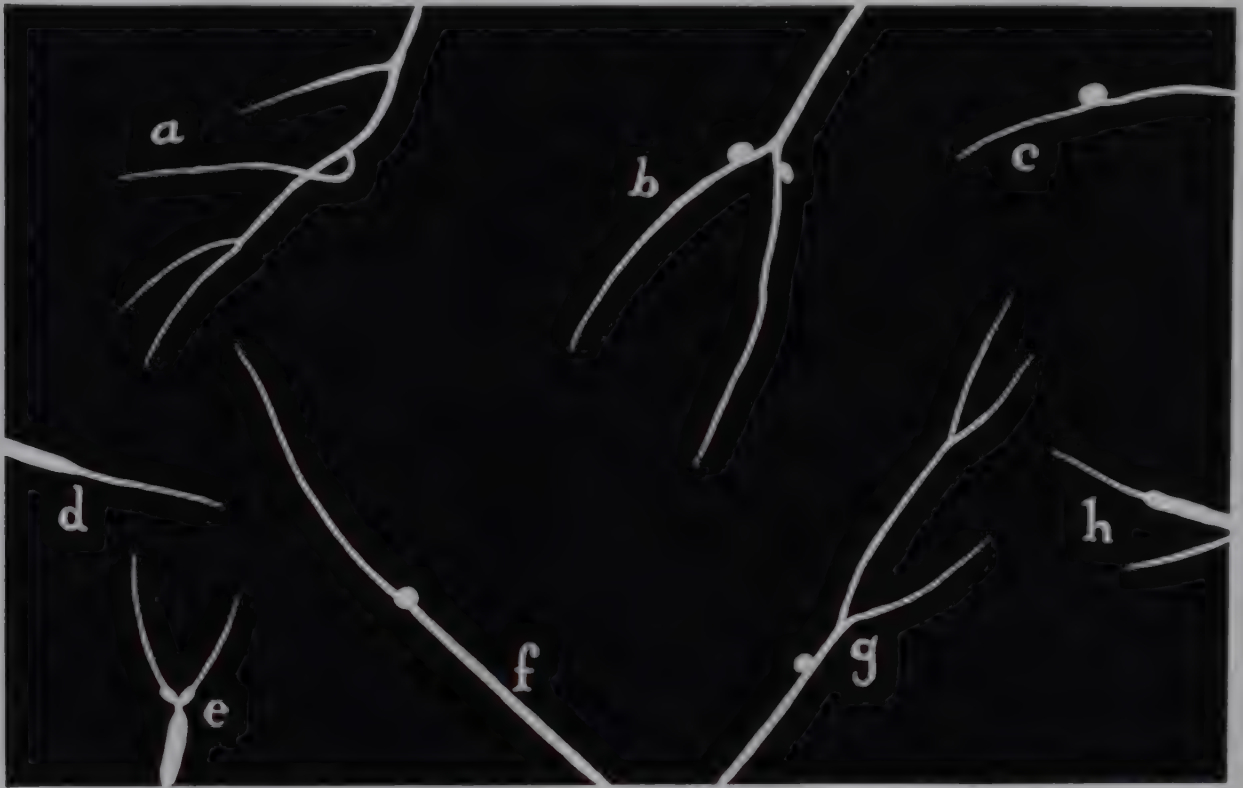


FIG. 154. Diagram of nerve bifurcations, showing branchings, abrupt ending of myelin sheaths, and Koeppe's so-called neurofibromata.

the surface of the parallelepiped appears homogeneous, giving no hint of its structure or of the presence of layers.* Actually, the gray surface of the parallelepiped represents Bowman's membrane because the overlying epithelium is practically transparent. However, in optic section, the anterior surface is seen to be formed by a double line, delineating the precorneal film and Bowman's membrane. The dark space between these two lines must be considered as representing the epithelium. It appears dark because it is relatively less respersive and relucant than the other layers of the cornea. When the anterior surface of the cornea is observed in the zone of specular reflection, the corpuscular elements of the tear film and meibomian secretion can be seen.

* Students are cautioned not to mistake the catoptric image of the lashes, formed on the anterior surface of the parallelepiped, for pathologic changes when examining the upper parts of the cornea in direct focal illumination.

THE POSTERIOR SURFACE

The further edge (Fig. 68, *fb*) of the posterior face of the parallelepiped is not as sharply delineated as the near edge, *eg*. Less light is reflected from the posterior surface, since the difference in the indices of refraction between the cornea and aqueous is less than that between air and the cornea. Study of the posterior face in the zone of specular reflection affords details of the endothelial cells, which appear as a characteristic mosaic. In this mosaic, in adults, dark circular concave spots are seen at the periphery of the cornea. These spots have been identified by Vogt as the Hassall-Henle bodies (warts) of Descemet's membrane (Fig. 155). The membrane itself, unless altered by pathologic changes, cannot be visualized.

OPTIC SECTION

Precorneal Film Layer. As the width of the slit is narrowed, the width of the slit beam correspondingly decreases (page 78). When such a narrow beam traverses the cornea, a veritable section is obtained. The importance of this optic section cannot be overemphasized, because its use justified application of the term "histology of the living eye" to biomicroscopy. Not only does it allow visualization of "minutiae," but it is of paramount importance in localization. When the narrow beam with a wide angle is used, it may be seen that at the point where the light strikes the cornea (Fig. 152 B), a double line is formed. The superficial or anterior one ("impact" or "incisive" line) represents the precorneal film. The precorneal film has not, until lately, received the attention it deserves.

According to Rollet²⁵² the thin layer of fluid should be individualized as a "*liquid precorneal layer*." This liquid precorneal layer is formed not only from the tear fluid, but also from secretions of the conjunctivo-palpebral glands of Manz, Krause, Henle, the tarsal glands, and the like. It has a complex mucolacrimal origin with a peculiar physicochemical composition, which not only has bactericidal properties, but also important properties of viscosity, cohesion, and resistance to evaporation, due chiefly to the presence

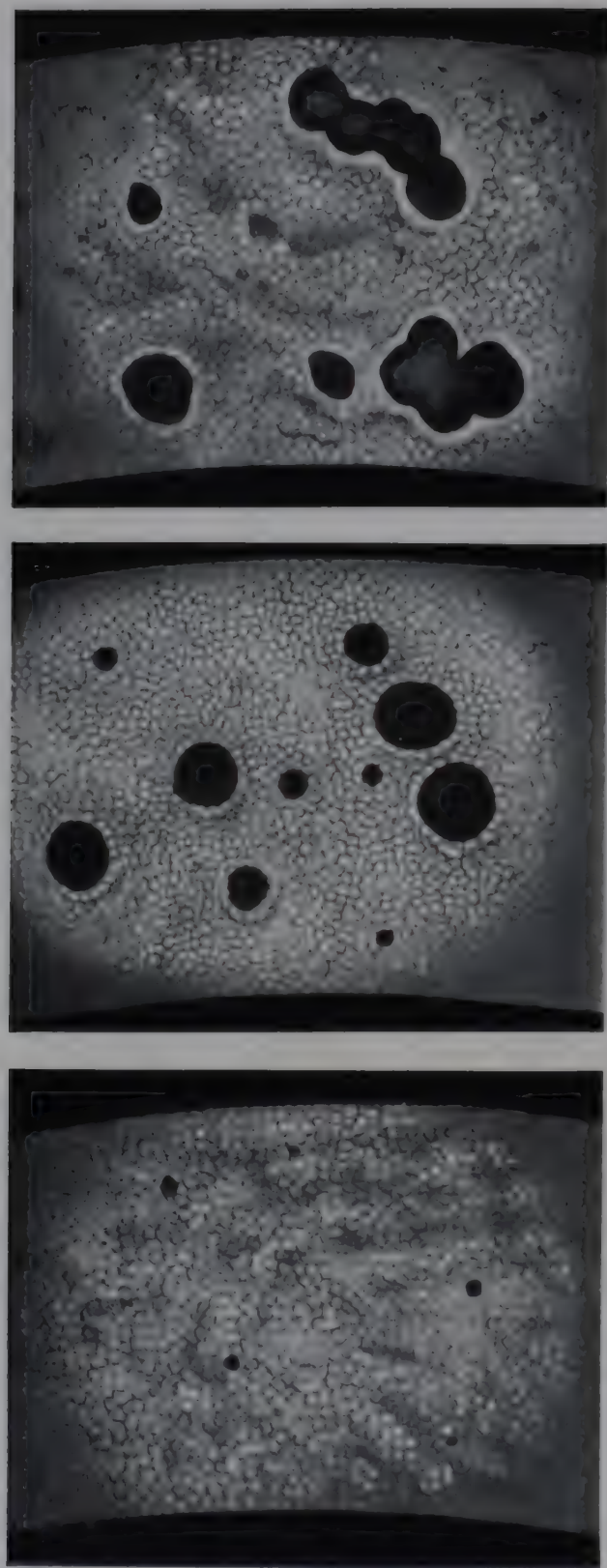


FIG. 155. Hassall-Henle bodies (Vogt). A. Amorphous (normal) appearance of the endothelium at limbus in specular reflection. Small black dots represent the Hassall-Henle bodies. B. Presenile appearance under higher power showing the crater-like dark Hassall-Henle bodies. C. Senile appearance. (About 60 X.)

of protein and lipoidal substances. Small air bubbles, which may form in the film, result from blinking movements of the eyelids.*

The importance of this layer is best illustrated by the blinking reflex: when the liquid layer becomes too thin because of evaporation, this reflex is stimulated. Pathologic disturbances in the precorneal film may affect the normal health of the corneal epithelium and cause changes. (See keratoconjunctivitis sicca, page 470.)

This layer can best be seen at the point where the lower eyelid touches the eyeball, especially when the gaze is directed slightly upward.† Here (Plate XVII, fig. 2) the precorneal film layer actually leaves the cornea and projects forward like the prow of a boat (*prow-line*) (Plate VIII, fig. 1). This is due to the fact that the tear fluid gravitates toward the eyelid margin and forms a pool. A drop of fluorescein instilled into the conjunctival sac colors the precorneal film a vivid green. The normal corneal epithelium is not stained by fluorescein, unless it is pathologically altered.

Epithelial Layer. The dark space beneath the precorneal film line must be considered to represent the thickness of the epithelial layer (Figs. 69, 152 B); it appears dark because the epithelium is relatively homogeneous and nonrelucant. This dark space widens as the periphery of the cornea is approached, showing that the corneal epithelium thickens as it approaches the limbus.

Bowman's Zone. Immediately behind this dark space the relucency of the corneal parenchyma begins, the top surface of which forms the second line and must be regarded as a part of Bowman's membrane, except at the limbus where it fuses with the superficial

* It has been shown that directly adherent to the corneal epithelium, there is a firmly fixed liquid layer which by interference studies (reflectography) is proved to be present even when the tear film layer itself has evaporated. It appears to have a close chemical relationship to the corneal epithelium and to play a considerable part in the vital gaseous interchange which takes place between the cornea and the atmosphere. Overlying this fixed liquid layer the film of tears acts as a buffer medium, influencing the gaseous interchanges. When the eyelids are kept open rapid evaporation of the tears occurs but the deeper liquid remains constantly fixed to the epithelium.

According to Fischer⁸⁶ pathologic states of the cornea, such as edema of the deeper epithelial cells, parenchymatous keratitis, glaucoma and the first stages of keratomalacia, are attended by quicker drying of the cornea than is seen in the normal eye when the eyelids are kept open, because of absence of the fixed liquid or humid layer.

† When examining the lower limbal regions, one will generally find it advantageous to retract the lower eyelid away from the eyeball.

PLATE XVII

FIG. 1. Corneal nerves coming from limbus.

FIG. 2. Optic section at limbus showing (*a*) precorneal film line (prow-line) at eyelid margin; (*b*) relucet wedge of Bowman's membrane at limbus (limbal spur); and (*c*) hour-glass type of arcus senilis.

FIG. 3. Normal cornea. Vital staining with polychrome methylene blue. Part of the epithelium stains as large blue cells with a dark center while parenchymal cells stain reddish. (After Knüsel and Vonwiller.)

FIG. 4. Vital staining with polychrome methylene blue of the cornea in direct focal illumination and retro-illumination. (After Knüsel and Vonwiller.)

FIG. 5. High power view of Figure 4. Retro-illumination.

FIG. 6. Vital staining of the corneal limbus showing corneal nerves, end organs, and punctate staining of cells. Vascular arcades are red in color. (After Knüsel and Vonwiller.)



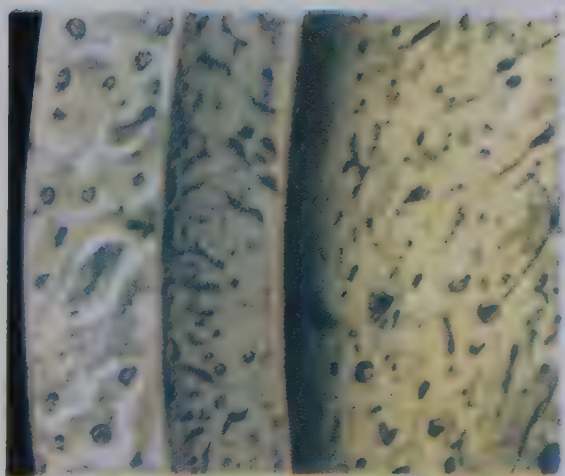
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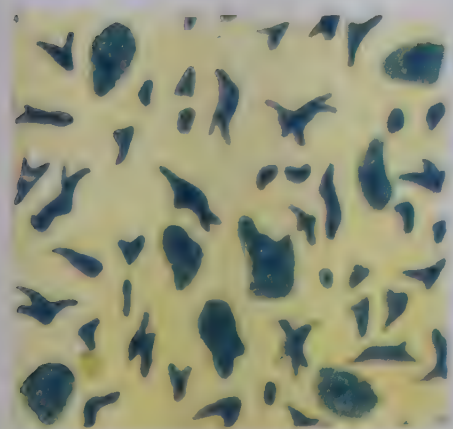
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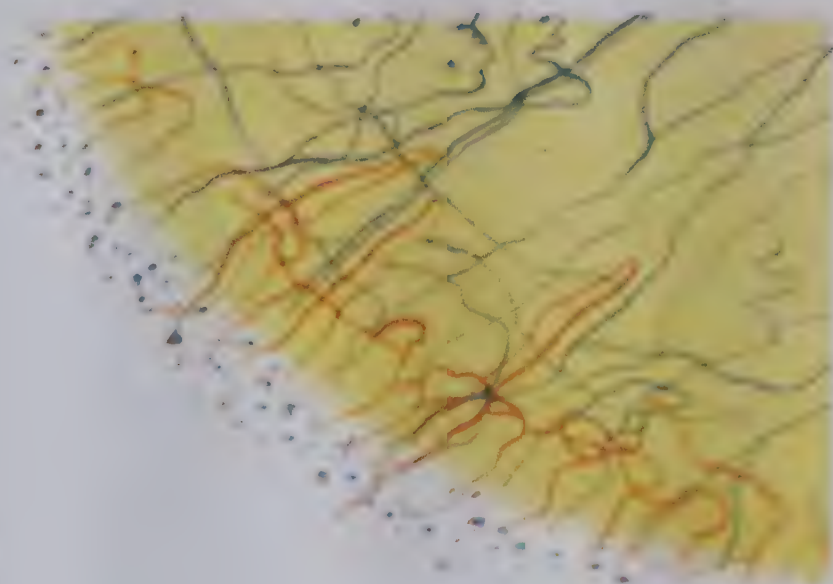
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6

limbal spur. It is only in altered conditions that this line becomes much more relucant than the subjacent parts of the section. The relucant line of Bowman's membrane ends near the limbus to join



FIG. 156. Droplets at limbus (retro-illumination). Physiologic bedewing of the epithelium extending somewhat axially to the limbal spur and perilimbal vascular loops.

the apex of a narrow wedge of relucant tissue, which is faintly seen by sclerotic scatter. This wedge which widens as it disappears into the sclera, has been named "the relucant superficial limbal spur" by Graves (Plate II, figs. 3, 4). The limbal vascular arcades are situated anteriorly to it.

In optic section, as in the case of the parallelepiped, the internal specularity of the cornea begins to appear as the zones of specular reflection of the corneal surfaces (Fig. 96) are approached. The line indicating the posterior face in optic section is slightly more relucant than the stroma. Another feature, occurring in the normal cornea, has been described by Vogt. This consists of a delicate edema (bedewing) of the corneal epithelium, seen in retro-illumination at the limbus (Fig. 156). The droplets are smaller than those usually observed in pathologic states. Vogt attributes their presence to an imbibition of the nutritive fluids from the neighboring capillary loops (Plate II, figs. 3 and 4).*

Both the optic section and parallelepiped have the geometric form

* The presence of physiologic cells on the posterior corneal surface is discussed on page 434.

of a hyperboloid. However, owing to the curve of the cornea, the curvature and thickness of the optic section or parallelepiped vary, depending on the incidence of the slit beam. This distortion is particularly noticeable if the beam is directed across the nose to the temporal side of the opposite cornea. Not only does the optic section become more curved, but at the extreme (temporal) limbus its apparent thickness almost doubles. This distortion can be avoided by having the patient change his direction of gaze, or by narrowing the incident angle. In the latter case, when examining the temporal side of the cornea, the arm of the illuminating system should be on the temporal side.

VITAL STAINING OF THE CORNEA

Clinically, vital staining of the cornea is of only theoretic interest to the biomicroscopist. However, surface staining of the precorneal film line and superficial lesions, especially those marked by epithelial denudation, is of great practical importance. Originally, dye solutions were employed for superficial corneal staining in order to outline breaks in the corneal epithelium.²³⁸ This is usually accomplished by instillation of a weak aqueous solution of an aniline dye into the conjunctival sac. The most satisfactory staining agent is a 2 per cent solution of sodium fluorescein. Any suitable water-soluble aniline dye-stuff may be employed but some of these have received more attention than others, namely, methylene blue, polychrome methylene blue, mercurochrome, rose Bengal, azur ii. The usual practice is to permit the stain to act on the cornea with the eyelids closed for a few moments. This facilitates the spread and penetration of the staining agent. The excess is washed out of the conjunctival sac with boric acid solution.

Staining by this method occurs in states of devitalization (epitheliolysis or of solution of continuity of the corneal epithelium. A characteristic bright green color is produced in areas in which Bowman's membrane has been stained by the solution. In this way the extent of an abrasion or epithelial denudation is outlined clearly. Ordinarily, normal epithelium does not stain, but changes due to

cocainization may cause staining (page 148, Vital Staining of Conjunctiva).

Coloring of the tear fluid results in tinting of the precorneal film layer, provided the dye is not washed out of the conjunctival sac. This causes the surface of the corneal parallelepiped to appear green; while in optic section, the green precorneal film layer is seen as a distinct line separated from the relucant underlying stroma by a dark band representing the nonresponsive, nonrelucant epithelium. However, owing to movements of the eyelids, the dye solution is rapidly washed from the epithelial surface, leaving irregular, mottled, unstained areas, which may be misinterpreted as pathologic alterations. Consequently, when making prolonged observations, repeated instillations of the dye solution are advisable.

Staining of the precorneal film line is of great value in demonstrating a surface irregularity, as well as a change in thickness of the epithelium itself. This is determined by variation in the width of the dark zone between the green-tinted precorneal film line and the relucant underlying Bowman's zone.

The presence of mucus or desquamated epithelial cells on the corneal epithelium may be demonstrated by staining or by interference with the tinted precorneal film line.

Although it has previously been stated that normal epithelium does not ordinarily stain, experimental staining of the epithelium, some of the parenchymal cells, and the nerve fibers is possible. Cocainization enhances this effect. Considerable work on the subject has been done by Knüsel and Vonwiller, Gallemaerts, Kleefeld, Marx, and Rados. It should be stressed that vital staining with some of the aniline dyes is not recommended for ordinary clinical practice, since the incautious or inexperienced use of such agents may result in injury. The action of the staining solution may be painful and the ensuing discoloration may last for a long time. Consequently, it is safer to use weak aqueous solutions (varying from 0.1 to 0.5 per cent) and repeat the instillations at varying intervals until the desired staining effect is obtained. In the event of severe pain, the instillations should be terminated and a suitable anesthetic instilled

or a bandage applied to the eye. Gallemaerts¹¹⁴ found that 0.4 per cent Nile blue sulfate solution, 0.4 per cent methylene blue, and 0.5 per cent azur ii were the best staining agents. One per cent rose Bengal solution has been recommended by Kleefeld.¹⁶⁷

Knüsel and Vonwiller,¹⁷⁰ using methylene blue, were able to obtain a superficial tinting of isolated groups of cells similar to the conjunctival cells, that is, large, round or polygonal cells with densely staining nuclei.

Overstained cells can be seen more distinctly in retro-illumination. Weakly stained cells lose dye quickly in direct focal illumination and can be seen only as shadows in retro-illumination. Desquamated epithelial cells stain partly or completely like scales.

In the presence of such pathologic changes as erosion, ulcer corneae or chronic iridocyclitis, the epithelial cells stain more quickly and easily. After corneal abrasion (e.g., removal of a foreign body) the borders of the defect stain diffusely blue. In the floor of the defect, parenchymal cells can be seen to take the stain. Occasionally, stained parenchymal and epithelial cells may be seen surrounding the lesion. Staining of the minute punctate lesions occurs in superficial punctate keratitis. Staining of the swollen epithelial cells is seen in sclerosing keratitis both in the involved areas and in the neighboring conjunctival areas. In erosion, if only the superficial epithelial cells are destroyed, the deeper cells are stained and are enlarged because of swelling. These larger cells are not seen in histologic preparations, possibly because of fixational shrinkage.

The parenchymal cells of the normal cornea can be stained by repeated instillations of polychrome methylene blue solution. In diseased conditions the parenchymal cells stain more readily. In the parallelepiped, polychrome methylene blue staining (Plate XVII, fig. 3) shows parenchymal cells as pinkish red stars. It should be noted that only an occasional cell stains by this technique. The dye penetrates only to the anterior middle layers of the cornea. With methylene blue, the parenchymal cells stain more deeply and more intensely blue than the larger, plumper epithelial cells. They appear stellate in shape. No nuclei can be discerned in the deep-lying

parenchymal cells. Retro-illumination renders localization and identification of the cells difficult because they appear mixed together (Plate XVII, figs. 4, 5). Differentiation between epithelial and parenchymal nerve endings is not easy. The parenchymal nerve filaments are clearly seen because of the peculiar affinity which nerve tissue has for methylene blue. As a matter of fact these structures sometimes may be the first to take the stain. It is impossible to stain Bowman's membrane unless the epithelium is denuded.

Fromm and Groenouw¹⁰⁰ showed that it was possible to obtain deep staining of the cornea by a solution of fluorescein instilled in the conjunctival sac, provided the dye was allowed to act for a considerable period. Also, von Hippel³³⁰ found that parenchymal staining follows when endothelial alterations are present. In this way parenchymal infiltrations and even keratic precipitates stain green.

In the region of the limbus, methylene blue stains a complex arrangement of branching nerve fibers of varying sizes, and the small corpuscular end organs at the termination of some of the fibrillae (Plate XVII, fig. 6). The larger stems of the corneal nerves lie more superficially than the terminal branches which penetrate the parenchyma. The superficial paramarginal nerve plexus is characterized by dichotomously branching nerve threads, which enter the conjunctiva. The smaller branches pass to their terminations in the parenchyma. Nerve fibers coming from conjunctival fibrillae also enter the parenchyma at the limbus.

Experimentally, 1 per cent osmic acid solution, after careful topical application to the surface of a leukoma, has been employed to stain cells containing fatty material dark brown to black, which contrasts with the nonaffected yellowish cells of the background.

Chapter Eleven

DEVELOPMENTAL ANOMALIES OF THE CORNEA

CONGENITAL UNIFORM DIMINUTION OF TRANSPARENCY

THIS condition was first described by Koeppe,¹⁷⁶ who observed it in eight cases. Vito also described three cases. The diagnosis can be made only with the biomicroscope since there are no gross signs of the condition, except diminution of vision. With the biomicroscope, the opalescence of the parallelepiped is seen to be exaggerated, indicating a peculiar, uniform and diffuse increase in corneal relucency. Otherwise, no definite alterations in the cornea are seen.

CHANGES IN THE SIZE OF THE CORNEA

MICROCORNEA (ANTERIOR MICROPHTHALMOS)

In this condition, the diameter of the cornea usually measures less than 10 mm. In contrast to microphthalmos, the eye otherwise is normal, although in some cases glaucoma may develop. There seems to be a definite familial tendency in the occurrence of this anomaly. When it is uncomplicated, the biomicroscope reveals little of importance, except for an increase in corneal curvature, as indicated by the shape of the optic section. In microphthalmos, which is usually associated with other severe deformities (cerebral), the corneae are correspondingly small. In a case recently observed (Plate XVIII, fig. 5) there was bilateral involvement with embryotoxon. This patient later developed uveitis in both eyes with marked increase in intra-ocular tension.

MEGALOCORNEA (ANTERIOR MEGALOPHTHALMOS, KERATOMEGLALIA, CORNEA GLOBOSA)

Megalocornea is differentiated from buphthalmos (congenital glaucoma) by the fact that it is due to a primary overgrowth and not to a secondary distention. It is characterized by enlargement of the entire anterior segment. It is bilateral and hereditary. Unless complicated by dislocation of the lens, the intra-ocular tension is normal. With the biomicroscope, the corneal tissue remains normal, except for the occasional presence of embryotoxon. In several cases that I have seen it seemed as though there was some increase in corneal relucency. In addition, above and below, crescentic relucient grayish bands continuous with the limbus were seen; these seemed to be caused by stretching of the nontransparent limbal scleral tissue. These bands somewhat resembled arcus senilis.

In three cases of megalocornea occurring in siblings (aged 5, 10, and 17 years), such peripheral arclike opacities, wider above and below and continuous with the sclera, were seen to be superficially placed in Bowman's zone. The superficial limbal arcades extended to the central edge of the opacity. The impression was gained that this opacity represented stretching and opacification of the transitional limbal zone rather than embryotoxon (Plate XVIII, fig. 1).

The posterior face shows no folds or ruptures in Descemet's membrane. The anterior chamber is deep, and there may be associated iridodonesis. Varying degrees of atrophy of the iris may occur. In a survey made by Vail,³¹³ cataract developed in approximately 40 per cent of the reported cases. Because of the associated enlargement of the lens and zonule, he suggested the name "anterior megalophthalmos" for this condition. According to Sugar,²⁸⁷ gonioscopy of the chamber angle in a case of megalocornea showed that the ciliary body portion was covered by pigmented tissue which extended to the line of Schwalbe. The trabeculum was obscured.

PLATE XVIII

FIG. 1. Megalocornea. Arc-like areas of increased relucency above and below.

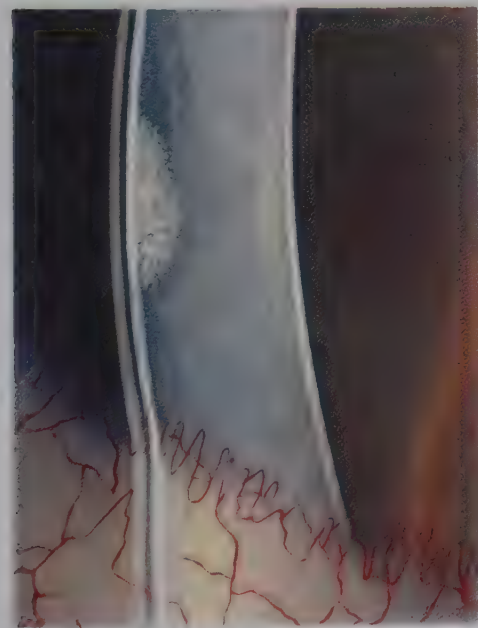
FIG. 2. Early arcus senilis. Direct focal illumination (optic section).

FIG. 3. Moderately advanced arcus senilis. Direct focal illumination (optic section).

FIG. 4. Advanced arcus, with dark lines (Vogt) and plexus formations. Direct focal illumination (optic section).

FIG. 5. Embryotoxon. Optic section through deposit in a case of microphthalmos.

FIG. 6. Pterygium. The optic section passing through the advancing infiltration in Bowman's zone.



BUPHTHALMOS (HYDROPHTHALMOS, OEIL DE BOEF OR
OX-EYE)

This condition is a form of infantile glaucoma, secondary to structural anomalies in the angle of the anterior chamber.* Although



FIG. 157. Ruptures in Descemet's membrane in buphthalmos (sclerotic scatter and proximal illumination). Relucent borders of tears or Haab's stripes.

it usually occurs bilaterally, many cases of unilateral buphthalmos have been reported. There is considerable evidence pointing to heredi-

* Barkan,²⁰ in a recent article, "Operation for congenital glaucoma," advocates early incision of the iridic angle (goniotomy) as a procedure to re-establish drainage from the anterior chamber into Schlemm's canal. He says: "If one can judge by the reduction of pressure that follows removal of the obstructing fetal tissue in those cases which are seen early, it must be assumed that Schlemm's canal is present. This assumption is confirmed by postoperative gonioscopy in the three aforementioned cases, and by the analysis of anatomic examinations of 84 specimens taken from eyes that had not been operated on, as reported by Anderson. He found that Schlemm's canal was present in 75 per cent of the earliest specimens. No sign of it was found in more than half of the specimens taken from children over 2½ years of age. He suggests that the canal becomes closed in the later stages as the result of distention of the eyeball and of increased intraocular pressure. The evidence obtained from anatomic specimens thus confirms the reason given on the basis of my gonioscopic examination for the effectiveness of incising the angle in early cases. It shows the importance of early diagnosis for successful operation and explains why the chances of success with this operation may be expected to diminish with the increasing age of the patient."

tary and familial influences in the production of this condition. The entire eyeball is enlarged, the anterior chamber is correspondingly deep, and the thinned sclera has a bluish color.



FIG. 158. Ruptures shown in Figure 157 as seen by direct focal illumination.

The effects of distention are revealed in the cornea at an early stage of the disease by generalized increase in corneal relucency and development of ruptures in Descemet's membrane. These ruptures may resemble fine-branched striae or they may be large and extensive; the margins of the ruptures are blurred, thus producing the so-called Haab's striae (Figs. 157, 158). In no other condition are ruptures so beautifully demonstrated. The optic section usually reveals marked thinning, especially in the central area of the cornea, and alterations in Bowman's membrane, that is, areas of increased relucency. Pigment deposits may be seen on the posterior corneal surface. When tension is high, conspicuous bedewing of the epithe-

lium occurs. In the later stages, the cornea may become completely opaque owing principally to changes in Bowman's zone. Biomicroscopy is of great importance in making an early differential diagnosis between megalocornea and buphthalmos.

ABNORMALITIES IN CORNEAL CURVATURES

CORNEA PLANA

Rübel²⁵⁶ described a rare bilateral flattening of the cornea, with a tendency to familial occurrence. In this condition the curvature of the cornea approaches that of the sclera. Because the sclera encroaches on the cornea, the cornea appears to be smaller than it actually is. Friede⁹¹ states that with the biomicroscope there seems to be an increase in density and in the number of the so-called corneal corpuscles. Barkan and Borley²⁰ reported three cases of familial cornea plana; in each case there was an indistinct opacity, extending from the margin of the sclerocorneal limbus into the flattened cornea (considered to be embryotoxon); the opacity was indistinct and of bluish white color. They believe that cornea plana is due to a maldevelopment occurring at about the fourth month of fetal life, when differentiation between the sclera and the stratified epithelium of the cornea occurs. In Swett's case,²⁹⁰ microscopic examination revealed many small linear grayish dots in the nebulous marginal zone at the limbus.

KERATOCONUS POSTICUS

This condition was described by Butler⁴³ in 1930. The posterior face of the cornea increases in curvature so that a thinning of the central corneal area results. In optic section it appears as though the anterior and posterior faces almost approximate each other. The curvature of the anterior face remains normal, and the parenchyma is clear. In contrast to keratoconus, it is not progressive.

POSTERIOR CRATER-LIKE FORMATIONS

Koepppe¹⁷⁸ described a rare congenital, bilateral anomaly, in which there are crater-like formations of the posterior corneal surface, ex-

hibiting an undulating irregularity. He compares it with the surface of the moon, as seen through a telescope. According to his description, this condition seems to resemble endothelial dystrophy. No further references to such an anomaly have been found in the literature.

CONGENITAL OPACITIES OF THE CORNEA

Congenital opacities of the cornea usually occur deep in the cornea or on its posterior face. They are difficult to evaluate because of their frequent association with other anomalies, such as microphthalmos, iris coloboma, congenital anterior synechiae, and congenital hyaline membranes. Therefore, in many instances it is not possible to determine whether they belong to the cornea per se or are part of a widespread congenital alteration. These opacities in the cornea may be part of an anomaly, primarily caused by a faulty differentiation of the "postendothelial" tissue, which in the early stages (12 mm. embryo in man) occupies the future anterior chamber (Ida Mann). This tissue is the anlage of the prepupillary membrane and iris stroma, and normally should lose its contact with the mesoblastic layer, forming the corneal endothelium. Failure of separation at this stage may account for certain posterior corneal anomalies. More extensive alterations, followed by fusion of these structures, may result in the so-called congenital anterior staphyloma. On the other hand, another school of thought attributes these opacities to intra-uterine inflammations. The presence of cellular deposits might be construed as indicative of intra-uterine ocular inflammation. The biomicroscope may be of great help in differentiating between these two pathogenetic factors.

CONGENITAL ZONULAR OPACITY OF THE CORNEA

Key¹⁶⁴ mentioned a rare congenital opacity of the cornea consisting of faint yellowish granules, which appeared white when viewed with the biomicroscope. This zone was marked off from the normal cornea by a faintly opaque line. The deposits were confined to the

anterior layers of the cornea. The surrounding cornea and even the posterior layers of the cornea behind the lesion were clear and transparent; there were no deposits on the posterior corneal face and no other observable ocular changes. Apparently this is one of the few recorded cases with biomicroscopic observation of a purely superficial congenital corneal anomaly.

CONGENITAL OPACITIES OF THE POSTERIOR CORNEAL SURFACE

These opacities are also known as congenital leukomas and may be familial. A porcelain-white or yellowish circular opacity occurs in the center or periphery of the posterior corneal face or deep within the corneal substance. The corneal opacity is hyaline in type and may exist either as an isolated finding or it may be associated with anterior iridic synechiae or defects in Descemet's membrane. Iridic tissue, emanating from the lesser arterial circle and traversing the anterior chamber, may be adherent to this opacity by delicate weblike expansions when the opacity is on the posterior face. In this case the opacity is generally grayish and translucent. This abnormality may be associated with microphthalmos with or without colobomas. As a rule the anterior chamber is shallow and the intraocular tension is normal or diminished; however, cases of atypical buphthalmos have been reported.

Mann²⁰⁹ described three cases in which a hyaline membrane formed on the posterior corneal surface, associated with adherent anterior iridic synechiae; in her cases, the opacities were situated at the periphery, occupying the same zone as embryotoxon. I have observed two cases which belong to this group of anomalies. These occurred in two girls (first cousins), 1½ and 6 years of age, respectively (Fig. 159). The family history was not significant. In each case there were circular opacities on the posterior corneal surfaces to which the expansions of anterior iridic synechiae were attached. At the site of attachment of the synechiae to the posterior corneal face, the endothelium, as seen by specular reflection, was defective. In the youngest, the right eye was microphthalmic and the mem-



FIG. 159. Hyaline membrane on posterior corneal surface in a case of microphthalmos associated with anterior iritic membranes and syndactylia.



FIG. 160. Embryotoxon of the cornea. A white streak situated in the deeper layers of the cornea in a child (diffuse illumination). (Courtesy of Dr. F. Bloch.)

brane covered the entire posterior face of the cornea. The lenses were clear and intra-ocular tension was normal. Both patients had partial syndactyilia of the fingers and toes.²⁷

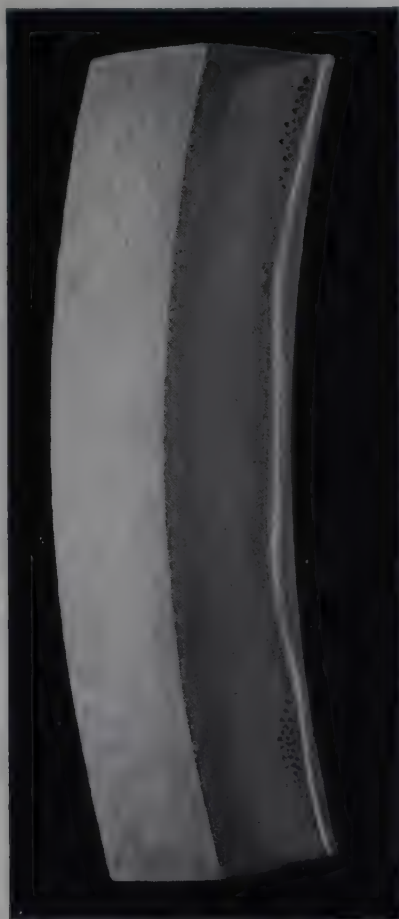


FIG. 161. The streak shown in Figure 160 as seen in parallelepiped (direct focal illumination).

EMBRYOTOXON

Embryotoxon is a counterpart of arcus senilis which develops either in fetal life or shortly after birth. It consists of an annular (complete or incomplete) or linear opacity involving the periphery of the cornea in its deeper layers. In the annular form, unlike arcus senilis, there is no clear ring of corneal tissue between it and the limbus; the opacity extends to the sclera without interruption. This condition seems to be hereditary (Landesberg¹⁸⁸) (Figs. 160, 161). Peters²³⁷ described a case of embryotoxon associated with blue sclerotics. Embryotoxon may also accompany megalocornea. In the case of bilateral microphthalmos which I have seen, a peripheral ring of embryotoxon at the limbus was present (Plate XVIII, fig. 5). The deposit extended through the deeper corneal layers.

Chapter Twelve

SENILE DEGENERATIONS, DYSTROPHIES, AND PIGMENTATIONS OF THE CORNEA

THE problem of differentiation between the physiologic changes of senescence and the pathologic degenerations which may be seen at any age has led to conflicting classifications of these conditions. It is known that the cornea tends to flatten somewhat and to become thinner with age. To some extent there is a diffuse increase in reluctancy probably associated with a slight rise in refractive index owing to senile sclerotic condensation of the parenchyma. Conditions which are usually peculiar to age may be seen in young adults, and may be due either to congenital lack of resistance of the tissue (abiotrophy) or to intercurrent disease. Primarily there appears to be an absence of signs of inflammation in all these degenerations and dystrophies.

The most frequent senile changes seen in the cornea are arcus senilis and Hassall-Henle bodies.

SENILE DEGENERATIONS

ARCUS SENILIS (GERONTOXON, ARCUS LIPOIDES, ARCUS PINGUICULUS)

This is a form of lipoid deposition in the peripheral cornea, associated with senescence; it appears as opaque grayish white arcs, usually beginning in the lower or upper portions of the cornea. In later years these arcs may fuse to form a complete circle, which, however, is separated from the limbus by a clear ring of superficial corneal tissue.

While this condition is commonly seen in the aged, it may occur early in life and is then termed *arcus juvenilis* or *presenilis*. Very often, in the third decade, the biomicroscope reveals increase of relucency of the peripheral portions of the cornea, consisting of groups of small whitish dots in the peripheral parenchyma. This cannot be seen with the unaided eye.

Commonly, the beginning of *arcus senilis* is seen in the lower limbus as a curved line of small, grayish, superficial or deep opacities, separated from the limbus by a zone of clear superficial corneal tissue (Plate XVIII, fig. 2). This so-called lucid interval (Vogt) measures from 0.2 to 0.3 mm. in the senile cases, but in juvenile *arcus* the interval may be so narrow that the opacity appears close to the limbus.

In most cases, senile individuals develop both upper and lower arcs which may fuse to form a complete circle. The deposits are usually broader and more prominent above and below than laterally, and the axial or central border is less sharply defined than the peripheral edge. Vogt described an instance in which double concentric ring formation occurred.

In marked *arcus*, the lucid interval sometimes appears to be depressed because the higher reflectivity of the adjoining scleral tissue and *arcus* causes an illusion of increased depth. This impression may be corrected by study with the optic section and fluorescein staining. Biomicroscopic section reveals that the infiltration consists of fine grayish dots varying in size and shape. Although in most instances, this appears first in the deeper parenchyma, in many cases it may appear in both the deep and the superficial layers either simultaneously or consecutively, or in one or the other alone.

When the deposit forms first in the deeper parenchyma, the subsequent anterior deposit is seen to develop directly over it. The two zones of opacity, in the beginning, are separated by a clear middle zone. As the condition progresses, the infiltrations may approach one another, sometimes producing an hour-glass appearance (Plate XVIII, fig. 3), but the deposit in the middle parenchyma is never dense.

The superficial deposition extends peripherally only as far as the termination of Bowman's membrane, at the superficial limbal spur, thus leaving a clear (lucid) interval, owing to the fact that the superficial limbal spur is thin and transparent where it joins Bowman's membrane. As the spur widens to join with the scleral tissue, it becomes slightly opaque, as is demonstrable in biomicroscopic section. As Graves¹³¹ pointed out there is frequently a tonguelike peripheral prolongation of the anterior zone behind but separated from the scleral spur (Plate XVIII, fig. 3).

Generally, at its termination there is an increase of relucency of Bowman's membrane itself over the area of superficial infiltration. In the deeper layers, the deposition extends without interruption into the sclera (Plate XVIII, fig. 4). Vogt³²⁰ described dark lines and plexus formations in advanced arcus. This occurs usually as sharply defined, dark, crossing lines which traverse the arcus obliquely or parallel to the surface. These lines may be from 20 to 50 microns thick. Frequently, their margins have fine serrations so that they appear to be *torn*, especially in the more superficially placed lines. Darker lines may occur in either superficial or deep zones and sometimes in the middle parenchyma. They may be quite prominent on the temporal side.

These lines appear to stop at the lucid interval and then to reappear in the sclera. They may extend up from the sclera, to be lost in the lower lucid interval, then picked up again in the denser portion of the gerontoxon, to be gradually lost in the clear portions of the cornea, axially.

Vogt considered that these lines represent preformed tissue spaces filled with fluid, and bear a strong resemblance to the tissue clefts seen in parenchymatous keratitis or compression of the cornea. They must be differentiated from the shadow lines seen in cases of folding of Descemet's membrane, or from the shadows of vessels at the limbus (the shadow moves with the beam) or even from the shadow-tracks caused by particles of dirt in the slit.

Versé³¹⁸ experimentally produced lipid arcs in rabbit corneas by feeding cholesterol.

HASSALL-HENLE BODIES

Hassall-Henle bodies (warts of Descemet's membrane) (Fig. 155) are constant biomicroscopic findings in all individuals over 20 years of age. They can be seen only in the zone of specular reflection of the posterior corneal surface, where they appear as dark, round spaces, in the golden endothelial mosaic, at times giving a false impression of holes or pits. This incorrect interpretation was rectified by Vogt,³²² who described them as wartlike protuberances rather than holes of Descemet's membrane. He estimated their diameter to be from 0.07 to 0.08 mm. and described them as projecting posteriorly between the endothelial cells. He identified them as Hassall-Henle bodies seen histologically.

PTERYGIUM

Pterygium (*πτέρυξ*, wing) is caused by the growth or extension of the bulbar conjunctiva on the cornea. It is shaped like a truncated pyramid with its apex toward the cornea. Although the main body of this structure is conjunctival, modern opinion believes that it is initiated from a degenerative process beginning in the cornea. However, after a loss of corneal substance (e.g., ulcer), an adhesion between the conjunctiva and the cornea may result in a "false pterygium." A false pterygium may occur at any point on the corneal circumference, whereas true pterygium is always found in the exposed interpalpebral zone.*

In biomicroscopic study of pterygium, our interest is mainly with the cornea, where the initial changes are seen. The first change observed is the presence of small grayish opacities in the cornea near the limbus in Bowman's zone. As the conjunctiva encroaches over the cornea, these opacities (which may become confluent) always precede the apex of the pterygium in its course toward the center of the cornea. In a fully developed pterygium, this grayish zone forms a flat rim around the raised apex. Between the grayish opacity and

* The original idea of Fuchs that a pterygium is always a form of conjunctival degeneration and that it is preceded by pinguecula is no longer accepted.

the apex, there is usually an area of clear cornea (Plate XVIII, fig 6). Schoeninger²⁰⁸ believes that the earliest opacities form at the place where the corneal nerves pierce Bowman's membrane. The vessels tend to run horizontally toward the apex. In optic section, the deeper portions have a dull yellowish tinge probably due to an increase in elastin in the subepithelial tissues. Histologically, Bowman's membrane becomes destroyed at these points and is replaced by fibroblastic tissue. At times small subepithelial cysts may be seen. The apex of the pterygium is covered by epithelium and in some instances may be grayish and somewhat avascular. In the progressively growing pterygium, the base may become quite fleshy and vascular.

Biomicroscopic examination is of importance in evaluating the rate of growth of a pterygium. Multiplication of the opacities preceding the apex, as well as extension of vessels into this zone, indicates a tendency toward rapid growth.

Cessation in growth is attended by decrease in size and flattening of the pterygium with attenuation of its vascularity. After surgical removal the corneal area usually shows subepithelial scarring with newly formed irregular vessels.

FUCHS' DIMPLES^{101, 108} (DELLEN, FACETS)

In 1911, Ernst Fuchs¹⁰⁸ described a condition in which small shallow superficial excavations occurred at the margins of the cornea at or near the limbus. These depressions follow interference with or obliteration of the limbal vessels associated with episcleral inflammations, tumors, cataract surgery, or prolonged use of cocaine. They may also occur in the aged in otherwise normal eyes. As a rule the dimples are asymptomatic and transient, rarely lasting more than forty-eight hours.

The dimples are small, usually ovular, and are situated parallel to and near the limbus, particularly on the temporal side. In optic section, a depression in Bowman's zone is usually seen with increased relucency in the floor of the dimple, appearing as a faint opacity (Fig. 162). In some rare instances, the dimples may be transparent.

In diffuse illumination, the depression may escape observation, if there is a light-colored blue iris as background.

In direct focal illumination, the dimple may throw a shadow on the iris because of its action as a concave lens.

In the original description by Ernst Fuchs, the steep part of the border was said to be situated at the limbus; but in a case subsequently described by Adalbert Fuchs¹⁰¹ the steep border was found toward the center of the cornea, the limbal edge sloping gradually. The latter author believes that these lesions are neuro-



FIG. 162. Fuchs' dimples or *Dellen*.

pathic in origin; this explanation is compatible with their rapid formation and disappearance and their sharp delimitation.

NONINFLAMMATORY CHANGES OF THE CORNEA OCCURRING IN DYSTROPHIES

Since the advent of the biomicroscope, our attention has often been called to the presence of small, whitish, dustlike opacities in or just below the epithelium. Such delicate changes may occur following the epithelial imbibition of commonly used medicaments (Fig. 163).^{*} At other times they are associated with infectious or allergic conjunctivitis (Fig. 128). Similar changes have been seen in other types of conjunctivitis (physical or chemical) and after contusions of the cornea. These changes in the cornea may be overlooked unless it is closely studied with the biomicroscope. In mild cases, a cornea

^{*} A case of a similar phenomenon due to hypersensitivity to pontocaine instilled in the conjunctival sac has been seen. A young man suffering from a mild acute conjunctivitis was given a solution of 0.25 per cent pontocaine to relieve discomfort. He instilled the drops several times a day, over a period of a week; the condition became aggravated with marked chemosis of the conjunctiva. Biomicroscopic examination showed the presence of many fine dots, diffusely sprinkled over the corneal surface. In optic section these dots were localized in the epithelium, forming an irregular design, just below the film line. Four days after discontinuance of the drug, all his symptoms abated and the whitish dots were no longer seen. A patch test gave a strongly positive reaction to pontocaine.

which is the seat of this type of epithelial change shows no areas which stain with fluorescein. In the more severe forms there may be marked photophobia and definite epithelial erosions. This may lead



FIG. 163. Superficial intra-epithelial dustlike changes twenty minutes after the instillation of a 4 per cent solution of cocaine. A. In direct focal illumination. B. In optic section. C. By retroillumination.

to the formation of surface flakes and filaments. It should be noted that these changes are much finer than those seen in superficial punctate keratitis or in rosacea keratitis, although thickening may be present in Bowman's zone.

Other epithelial changes similar to the alterations following the instillation of cocaine may also be seen (Fig. 163); among these are localized edema (bedewing) or desiccation occurring as irregular



FIG. 164. Epithelial and juxta-epithelial conditions. Inflammatory and kindred affections primarily epithelial. A,B, Fine punctate foci. C, Surface irregularities. C', Vacuoles. D,E,F, Thickening in Bowman's zone such as occurs in marginal ulcer. G,H, Intra-epithelial dots. J, Pyriform vacuoles. K, Punctate intra-epithelial refractile changes. L, Relucent epithelial changes. M, Vesicle. O, Textural disturbances of superficial stroma. N, Deep parenchymal changes. P, Folds of Descemet's membrane and precipitates. Q, Resulting relucent foci in superficial stroma with edema of overlying epithelium and superficial punctate gray specks. Lesions shown in Q and R may resolve into such lesions as those shown in T,U,V,W, which are similar to those seen in D,G,H. X, represents deep, associated changes in parenchyma in dendritic keratitis. Y,Z, Subepithelial changes and fissures as seen in dendritic keratitis. (Graves. In Diseases of the Eye. Berens, Ed. Courtesy of W. B. Saunders Company.)

linear designs or fine stipplings. These delicate changes may simulate those occurring in inflammatory conditions or trauma and in all likelihood represent a nonspecific reaction of the epithelium (Fig. 164); a neuropathic element may be involved. The state of the epithelium seems to be a sensitive index of corneal health.* Endothelial disease (as in Fuchs' dystrophy) may be followed by such epithelial change. Keratitic precipitates of long standing also may evoke similar epithelial reactions.

These changes occur in a more exaggerated form in the so-called hereditary and constitutional dystrophies of the cornea. They must be differentiated from the inflammatory types of superficial kerato-

* Any change in the transparency of the cornea is associated with a change in colloid properties leading to fluctuation in turgescence and consequent alteration of the index of refraction with increase in relucency.

conjunctivitis, keratitis neuroparalytica, and herpetic keratitis. The hereditary dystrophies are characterized by a peculiar symmetry of design, bilaterality, chronicity and, in most cases, by the absence of



FIG. 165



FIG. 166

FIG. 165. Epithelial dystrophy of Fuchs. Early stage. Superficial subepithelial punctate dots and beginning dystrophic changes in the endothelium.

FIG. 166. The superficial changes (Fig. 165) in optic section.

inflammatory (conjunctival and corneal) vascular response. However, inflammatory reaction may occur in the advanced stages of dystrophy due to secondary corneal alterations.

The endothelium, either alone or in conjunction with epithelial or

parenchymal alterations, may exhibit certain changes. This is well demonstrated in some of the corneal dystrophies in which a variety of endothelial changes occur; for example, wartlike changes, loss of endothelial cells, folds, and pigment deposits (Fig. 165; Plate XIX, figs. 2, 3).

By retro-illumination dewlike formations attributed to excrescences of Descemet's membrane may be seen, covered by powdery pigment ("cornea guttata" of Vogt). At other times, delicate striae may form in this zone.

In the parenchyma a host of alterations has been described, varying from isolated fine dots, granular or crumblike aggregations or dense nodules to lattice-like reticular formations, which may be either delicate threads or tubes (Figs. 173 to 180).

In keratoconus, a series of short parallel striae are commonly found in the parenchyma near the apex of the conus.

In lipin keratitis, the deposits of lipin material occur as small yellowish gray dots of varying size in the middle and deeper layers of the cornea. In other types, actual deposits of crystals (cholesterol or uric acid) may be seen in the form of glistening rods.

HEREDITARY AND CONSTITUTIONAL DYSTROPHIES OF THE CORNEA

There are a large variety of dystrophies, many of which seem to have a predilection for certain layers of the cornea. Some of these are confined to the epithelium (diffuse epithelial dystrophy, hereditary erosions) and others to the endothelium (cornea guttata); still others begin in one layer and later extend through the entire cornea (nodular, macular, and lattice dystrophy). A form which involves the endothelium and the epithelium (Fuchs' epithelial dystrophy), leaving the middle layers unaffected, is also seen.

Changes in the cornea, morphologically similar to the hereditary corneal dystrophies, are observed in bacterial or viral infections (herpes), nutritional disorders (vitamin deficiency: A, B₆), and trophic disorders (keratitis neuroparalytica) (page 488). Consequently, errors in diagnosis are frequent, especially in the early stages

PLATE XIX

FIG. 1. Fairly advanced stage of Fuchs' epithelial dystrophy. Epithelial changes consisting of vacuoles and bullae (by retro-illumination).

FIG. 2. Fuchs' epithelial dystrophy. Bullae and epithelial and endothelial alterations in parallelepiped (direct focal illumination).

FIG. 3. Fuchs' epithelial dystrophy. Same lesions (Figs. 1 and 2) viewed in optic section. Shadows extending backward from bullae are exaggerated.

FIG. 4. Secondary form of band-shaped marginal degeneration of the corneal periphery following chronic iritis. (Diffuse illumination.)

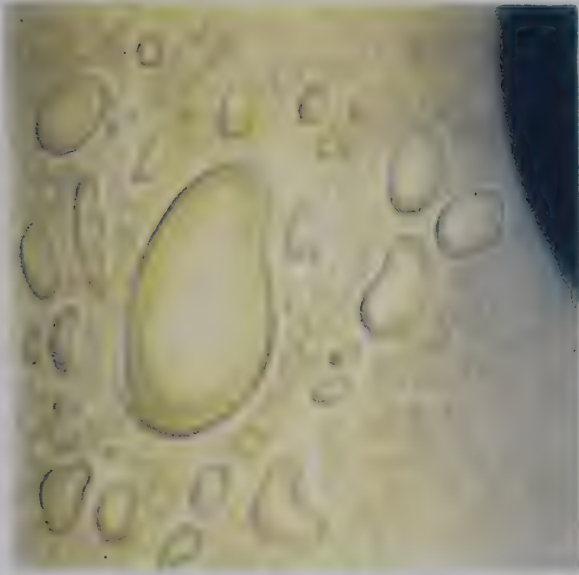
FIG. 5. High power view of same lesion (Fig. 4) in direct focal illumination (optic section) and by retro-illumination.

FIG. 6. Band-shaped keratitis (sclerotic scatter).

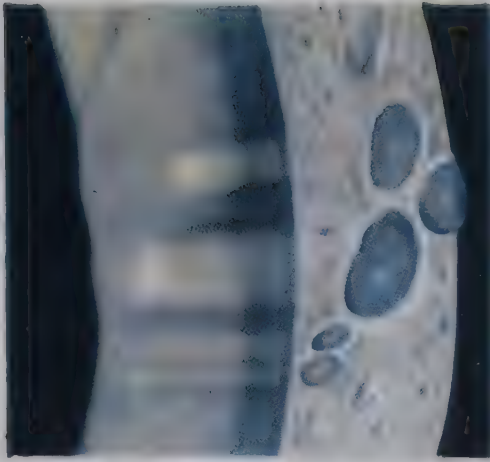
FIG. 7. Band-shaped keratitis. High power view of Figure 6. Note typical dark hole-like defects. The holes to the right appear by direct retro-illumination. (Direct focal illumination and retro-illumination.)

FIG. 8. Superficial crystalline corneal dystrophy. Appearance in direct focal illumination showing diffuse varicolored crystalline deposits.

FIG. 9. View of the crystalline deposits in Bowman's zone as seen in optic section of case shown in Figure 8.



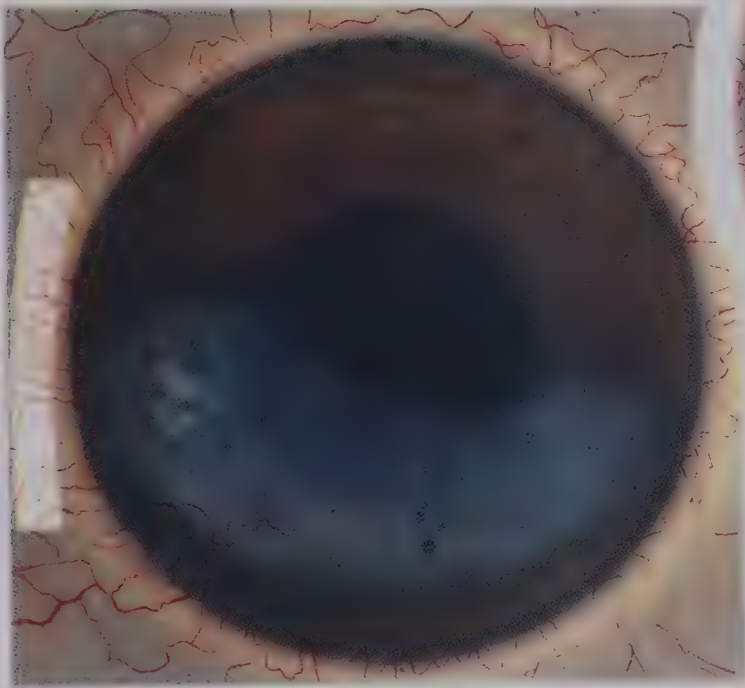
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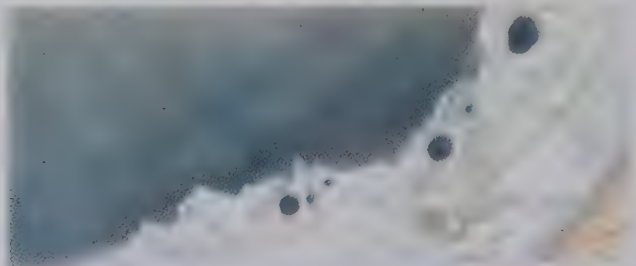


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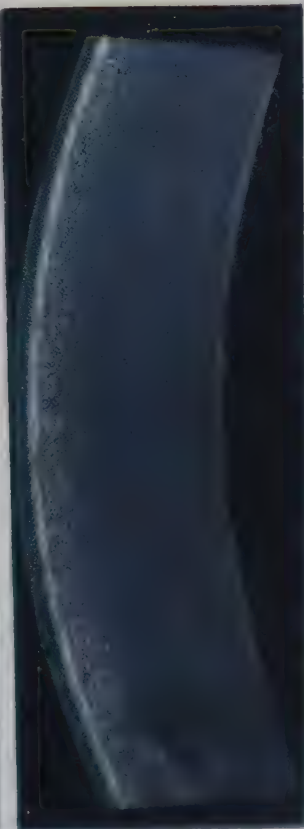
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of these dystrophies. However, a careful history, both personal and familial, and the symmetry and bilaterality of the lesions and their tendency to be chronic and progressive, establishes the true nature of these affections. In the light of recent work, in which many pedigrees have been studied, it is now known that there is a definite inheritance potential in the various corneal dystrophies. The majority of corneal dystrophies with opacities are now considered to be of dominant hereditary transmission. One type of familial dystrophy (spotted macular corneal dystrophy of Groenouw, type II; Fehr, type I) has been shown to be of a recessive character. Consanguinity is usually present in this form. Bücklers' ⁴⁰ investigations have shown that the familial corneal dystrophies must be considered from the point of view of heredity as well as that of clinical appearance.

Whether or not it is proper in the present state of our knowledge to consider such entities as lipin keratitis, keratoconus, limbal girdles, and primary bandform keratitis as hereditary or constitutional dystrophies may be questioned. Those interested in ophthalmic genetics show an increasing tendency (and with good reason), to so classify all purely noninflammatory or nontraumatic lesions.

The confusing terminology of older authors was due largely to the description of isolated cases, many of which were in intermediate stages of development. Some writers believe that the various types of corneal dystrophy are merely manifestations of the same parent disease (Fleischer). It was only after many cases had been studied biomicroscopically for a long period of time through the various stages of their development, and an analysis of the hereditary factors was made, that some light was shed on this problem. In the entire group of the so-called familial corneal dystrophies, Bücklers has found no instance in which there has been conversion or transformation of one distinct type into another.

The following classification (modified after Franceschetti and Streiff ⁹⁰) includes, in addition to the definitely hereditary dystrophies, certain forms of noninflammatory constitutional dystrophic corneal changes in which familial and hereditary factors have not been entirely established.

CLASSIFICATION OF HEREDITARY AND CONSTITUTIONAL DYSTROPHIES
OF THE CORNEA

- I. Epithelial dystrophies (hereditary)
 - A. Multiple recurring erosions (Franceschetti): dominant
 - B. Diffuse epithelial dystrophy (Pameijer, Meesmann and Wilke²²²): familial
 - C. Symmetric partial epithelial dystrophy (Franceschetti and Streiff)
 - D. Epithelial dystrophy of Fuchs: dominant
- II. Endothelial dystrophies
 - A. Cornea guttata (Vogt): dominant
 - B. Cornea guttata in association with epithelial dystrophy (Fuchs)
- III. Dystrophy of the entire cornea
 - A. Familial corneal dystrophies
 1. Nodular (Groenouw, type I, nodular, granular or crumb-like): dominant
 2. Macular (Fehr and Groenouw, type II): recessive
 3. Reticular, lattice-like, or grill-like (Biber, Haab and Dimmer): dominant
- IV. Circumscribed involvement of the corneal stroma (without primary epithelial or endothelial involvement)
 - A. Whorl opacity; superficial vortical pigmentation (Fleischer *)
 - B. Circular peripheral opacity (Biozzi and Lugli)
 - C. White rings of Coats — Bowman's zone
 - D. Keratitis nummularis (Dimmer) †
 - E. Cornea farinata (Vogt)
 - F. Superficial and deep "crocodile" shagreen (Vogt)
 - G. Primary lipin degeneration
 1. Dystrophia adiposa corneae
 2. Hurler's syndrome or gargoylism (dysostosis multiplex)
 3. Hereditary crystalline degeneration²⁷¹
 4. Embryotoxon
 - H. Dystrophy of keratoconus
 - I. Marginal dystrophy

* See corneal pigmentation, page 371.

† This entity is still considered an inflammatory keratitis by most authors and is described under "inflammations" (page 461).

- J. White limbus girdle
- K. Primary form of bandform keratitis
- L. Hyaline and calcareous degeneration

Epithelial Dystrophies

Multiple Recurring Erosions. Von Szily,^{331a} in 1900, described an hereditary affection of the corneal epithelium, characterized by multiple recurring erosions. The lesions are limited to the corneal epithelium; the stroma and the endothelium are not affected. In 1928, Franceschetti^{59a} reported a remarkable pedigree, illustrating dominant transmission of recurrent erosions in five generations. He cautions "that during the initial stages of the dominant type of corneal dystrophy (nodular or granular familial dystrophy), periods of irritation may occur from epithelial changes simulating recurrent corneal erosions."

Diffuse Epithelial Dystrophy (Pameijer).^{231a} Although Vogt described a rare form of diffuse epithelial keratitis occurring in a young individual, he made no mention of any familial tendency in his case. On the other hand, Pameijer observed the condition in several members of two consecutive generations, in which there was a development of minute epithelial vacuoles. The parenchyma and endothelium, as well as the corneal sensitivity, was unaffected.

Symmetric Partial Epithelial Dystrophy (Franceschetti and Streiff). In commenting on this variety of epithelial dystrophy, Franceschetti and Streiff⁹⁰ state: "We have observed two cases of a peculiar symmetrical dystrophy of the epithelium in which only the inferior nasal quadrant was affected. By retro-illumination the cornea appeared studded with opacities and minute erosions such as may be seen in the cornea when a small foreign body has been present under the upper lid; here and there the changes resemble those observed in herpetiform affections. In both cases the sensitivity was normal."

Epithelial Dystrophy of Fuchs. In 1910, Fuchs¹⁰⁷ described a bilateral corneal dystrophy occurring in adults, usually over 50

years of age, especially in females. Since his original report, many cases have been cited in the literature. This condition may be familial, as attested by its appearance in siblings. It is characterized by extensive



FIG. 167. Fuchs' epithelial dystrophy, more advanced, associated with endothelial changes.

epithelial and subepithelial changes. The changes begin with the formation of a fine epithelial bedewing in the central region of the cornea, which then spreads peripherally. Coalescence of these droplets results in vacuoles which rupture, leaving irregular areas of subepithelial opacification. As the subepithelial layers become more involved, opacities in the deeper layers follow (Figs. 165, 166, 167). Dark irregular lines at various levels in the parenchyma, resembling folds of Descemet's membrane and surrounded by pearly relucant areas, have been observed. Fine dots or gray striae may also be found in the parenchyma. Corneal sensitivity is markedly diminished in most cases and in some complete anesthesia is present. Owing to the chronic progress of the disease there is a gradual loss of vision. Of thirteen cases reported by Fuchs, three developed glaucoma. The formation of bullae may be accompanied by severe pain. In optic section

(in cases without ulceration) after the stage of initial edema, small whitish opacities are seen just below the film line of Bowman's zone. Many forms of opacities may be seen in the anterior portions of the section (Plate XIX, figs. 1, 2, 3). In severe and chronic cases, the entire cornea becomes opaque and large areas of superficial ulceration occur with extensive exfoliation of the corneal epithelium (Fig. 168). After ulceration, staining with fluorescein demonstrates gross superficial irregular areas of denudation.

Associated with this condition Kraupa¹⁷⁹ described endothelial changes, which, he stated, preceded the superficial epithelial changes.

Vogt, in 1921 and 1930,^{322, 326} independently described these early endothelial changes as resembling enlarged Henle warts and named the lesion "cornea guttata." Later, Friedenwald,⁹² Gifford,¹¹⁷ Dog-

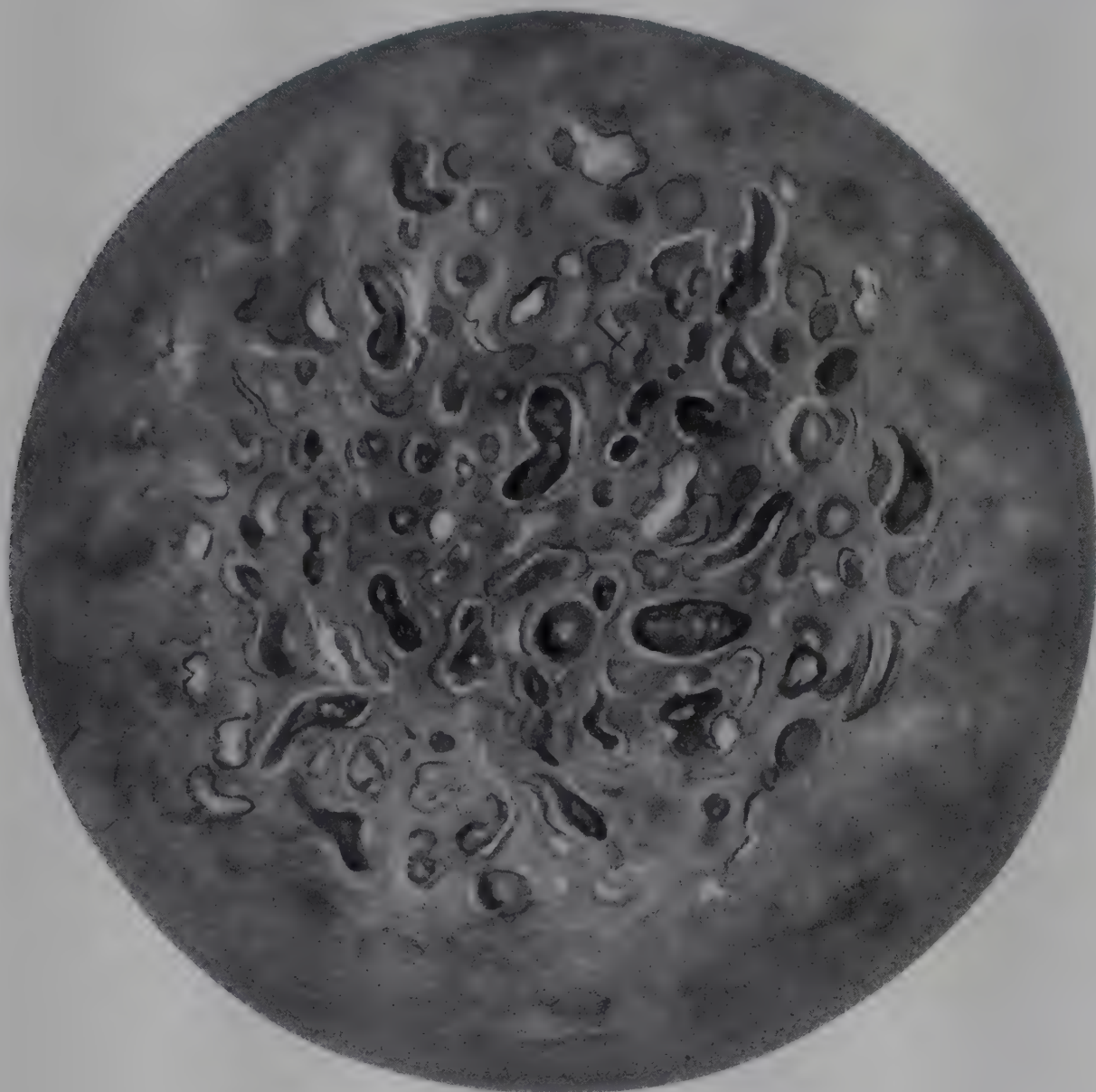


FIG. 168. Fuchs' epithelial dystrophy, late stage.

gart,⁶¹ and others contributed reports on this type of early endothelial dystrophy in Fuchs' epithelial dystrophy. The current opinion is that Fuchs' epithelial dystrophy (senile type) begins with endothelial changes, which are responsible for the later epithelial and parenchymatous degenerations.

Duke-Elder⁶⁸ states that "the two diseases (i.e., Fuchs' epithelial dystrophy and endothelial dystrophy or guttata) may be separated to the extent that the progress of the degenerative changes may be extremely slow, and the one may not develop into the other; but

the complete clinical picture appears to be first an endothelial degeneration allowing access of intra-ocular fluids in the cornea which is followed by the development of dystrophic changes in the epithelium and eventually by dystrophic changes in the substantia propria."

Theodore²⁹³ noted that in his series of congenital cases of endothelial dystrophy, no epithelial disturbances were seen, even in the oldest patient (77 years of age). This agrees with the report of a congenital case by Kraupa in a 70-year-old female. Theodore, following Kraupa, conjectures that there may be two types of endothelial dystrophy: (1) a senile degenerative type in which superimposed epithelial dystrophy may occur, and (2) a congenital fixed type which does not develop epithelial or parenchymal changes. Consequently, the latter type is described as a distinct entity.

Endothelial Dystrophy

Cornea Guttata. Since the invention of the biomicroscope, descriptions and reports of cornea guttata have been numerous. Koeppe¹⁷⁸ originally described a similar condition in 1916 and called the crater-like formations on the posterior corneal surface *Dellenbildungen*, concluding that the condition was congenital and due to an anomaly of development of the posterior face of the cornea.

The changes vary, evidently depending on their duration and type (i.e., congenital or senile). Graves¹²⁷ gives a classic description. In parallelepiped on the posterior face in the central areas, he saw "scattered lustrous glints" of a bronze color. These glints became more intense as the zone of specular reflection of the posterior corneal surface was approached. He attributed them to reflections coming from the rims or edges of rounded areas, which in themselves appear dark in the bright endothelial mosaic of specular reflection corresponding to but larger than the Henle warts normally seen in the periphery. These structures are convex posteriorly toward the anterior chamber. In addition, a fine pigment dust, seen also by retro-illumination, is usually present.

By direct or indirect retro-illumination, these areas also illustrate an example of refractile properties (Graves), causing "reversed illumination." This phenomenon depends on the refractile properties of pathologic deposits in or on the cornea. The lenslike action of convex-surfaced droplets, consisting of refractile material, is well known. In this connection, the reversal of illumination is caused by bending the light beam away from the point of incidence. Therefore, if the droplet is obliquely illuminated, a crescentic light halo is formed on the distal border, while the proximal border is left in comparative darkness. In addition to the refractile properties of the droplet the surfaces exhibit reflective properties, which also contribute to the concentration of the light beam on the distal border. Kirby¹⁶⁶ in 1925 and Gifford¹¹⁶ in 1926 reported cases of this type, which they called endothelial dystrophy of the cornea.

Histologically, Vogt³²⁷ as well as Goar¹¹⁹ found that Descemet's membrane actually forms excrescences pointing toward the anterior chamber; these were seen in areas either covered by a thinned atrophic endothelium or entirely denuded of cells. The nature of the rarer parenchymal changes varies from case to case. Vogt described an instance in which there was sudden widening of the optic section owing to the presence of a large vacuole in the corneal parenchyma causing secondary visual distortion. The vacuole was surrounded by a relucant area.

A case of a woman, 70 years of age, who first showed typical endothelial changes in both eyes, which were followed within two years by subepithelial degeneration, merits description (Fig. 167). Both corneae were relatively anesthetic. In the central areas, the typical glintlike reflections from the posterior face of the cornea were observed. Small groups of scattered pigment cells were seen on the endothelium. Anterior to these changes, the irregular lines in the parenchyma were seen. The characteristic punctate subepithelial opacities occurred in both eyes within two years.

Theodore²⁹³ described three cases of endothelial dystrophy — in a girl, her father, and her paternal grandmother, which emphasized the hereditary aspects of the disease (Figs. 169, 170, 171, 172). In

the case of the girl, opacities were noted at the age of three months. In all three cases (the youngest was also seen by me in consultation), the anterior corneal layers were normal. The epithelium, Bowman's

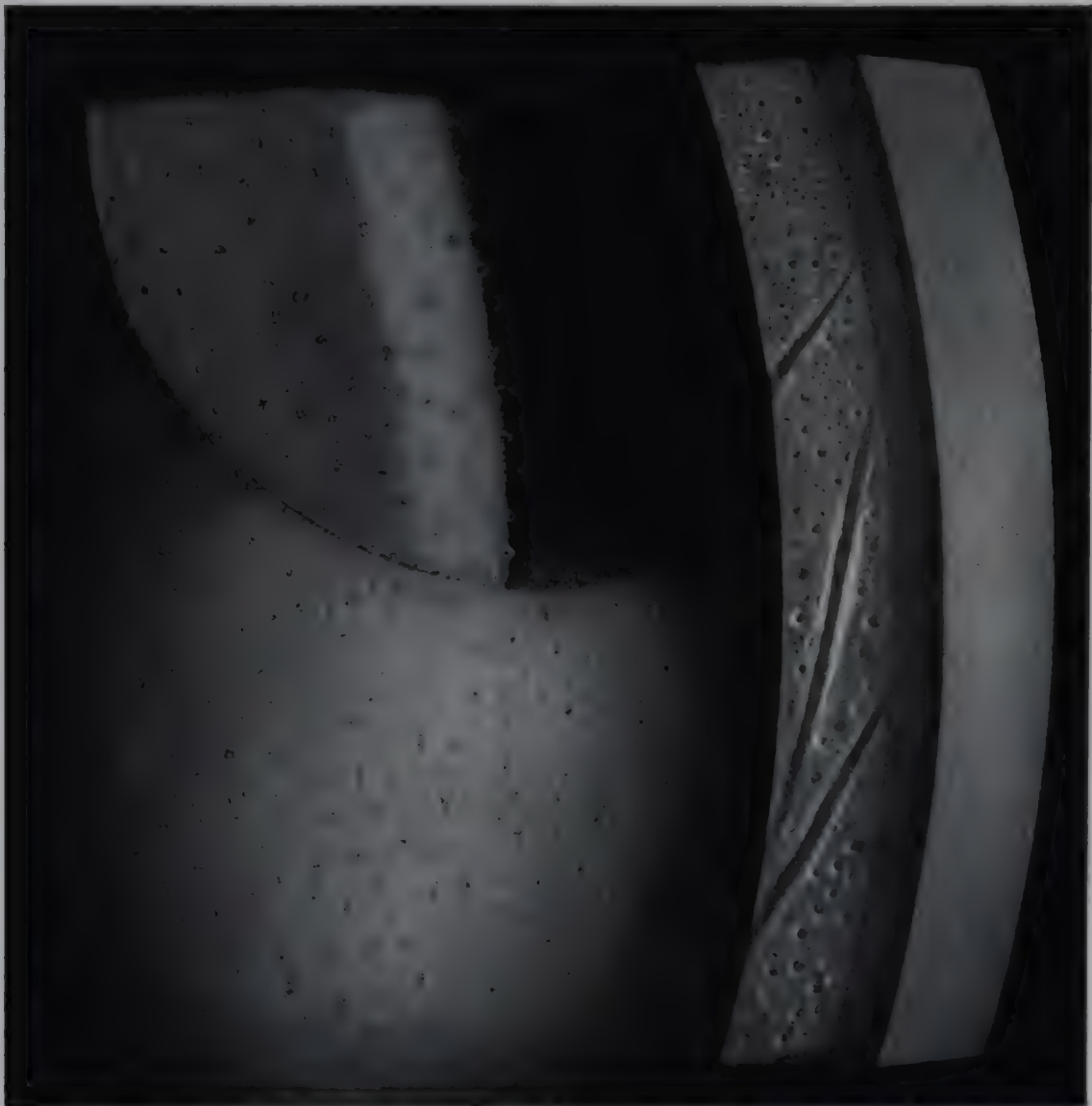


FIG. 169. Keratitis guttata showing folds of Descemet's membrane and endothelial alterations.

zone, and the anterior two thirds of the substantia propria were entirely normal. However, the deepest portions of the parenchyma showed a faint increase in relucency. The posterior face of the cornea was studded with large and small, irregular, round and elliptic clear structures, more numerous in the pupillary zone. These clear structures were nodules or thickened depressions rather than vacuoles. In optic section, the larger depressions had a definite anterior convexity. The posterior boundaries were not sharply outlined. The largest nodules measured 0.75 micron in diameter, the average being 0.2 micron. By retro-illumination the nodules appeared as empty spaces. The endothelial mosaic (specular reflection)



FIG. 170

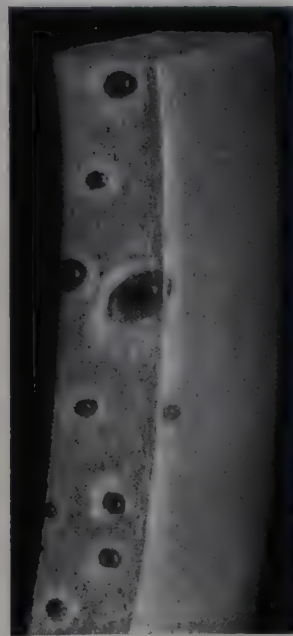


FIG. 171

FIG. 170. The appearance of the posterior corneal changes in specular reflection showing peculiar glintlike reflections. (After Theodore.)

FIG. 171. The appearance of the posterior corneal changes in direct focal illumination. (After Theodore.)



FIG. 172. Keratitis guttata showing endothelial changes in specular reflection.

was absent in the region of the nodule but normally present in other areas. In some places, however, the normal adjacent endothelium merged into the distorted portions, causing the characteristic "scattered lustrous glints," surrounding the dark area of the nodules denuded of epithelium. Only in the case of the grandmother was there considerable pigment deposition on the endothelium.

Dystrophy of the Entire Cornea

Familial Corneal Dystrophies. Under this heading are included several types of bilateral hereditary corneal degenerations. These changes usually appear at puberty but in rare instances have been observed in infancy. They run a long chronic course, gradually progressing so that severe visual damage may result.

When Groenouw¹³⁶ first described the nodular dystrophy which bears his name, he questioned the hereditary nature of this corneal disturbance. The genetic researches of Bücklers and others have since shown that the corneal dystrophies are of a definite familial type.

Modern ophthalmologic opinion concerning the classification of these dystrophies follows the lines laid down by Bachstez,⁷ Bücklers,⁴⁰ von der Heydt³²⁹ and Franceschetti and Streiff.⁹⁰ Three different types of familial corneal dystrophy are now distinguished: (1) *Nodular* type (*dystrophia corneae granulosa*): granular or crumblike (Bücklers) — dominant form of Groenouw's familial corneal dystrophy (type I). (2) *Macular* type: spotted (*dystrophia corneae maculosa*) — recessive form of Groenouw's familial corneal dystrophy (type II) — macular corneal dystrophy of Fehr. (3) *Lattice-like* type (Biber) (*dystrophia corneae reticulata*) — dominant reticular dystrophy (Dimmer) — grill-like keratitis (Haab).

These dystrophies must be distinguished from congenital opacities. The latter are found at birth, are nonprogressive and are frequently associated with other congenital alterations; whereas the former are bilateral, progressive, appear and develop well after birth, are not vascularized and have a hereditary background. Dystrophies occurring in the same family often reveal similar characteristics. Although Bücklers has shown that one form does not change into

Circumscribed Involvement of the Corneal Stroma (Without Primary Epithelial or Endothelial Involvement)

Circular Pevipberal Opacity. According to Biozzi and Lugli²⁹ this familial corneal affection resembles folds in Descemet's membrane and appears as a grayish white, linear, double-contoured streak localized in the posterior corneal layers. The opacity occurs as arcs concentric with the limbus and lying a short distance from it. The arcs may be joined, involving almost three fourths of the lower corneal circumference, or may be separated, or only one quadrant may be involved. The arcs have irregular outlines and vary in width from 0.04 to 0.15 micron.

White Ring of the Cornea (Coats). With the aid of biomicroscopy, this supposedly rare affection, originally described by Coats,⁴⁸ has been found to occur more commonly than was thought in the past.

The lesion consists of a small whitish gray ring, irregularly ovalar or circular in shape, usually near the periphery, but occasionally more centrally located (Fig. 183). High magnification discloses that these rings are composed of chalklike granules (varying in size from 0.5 to 1.0 mm.), situated in Bowman's zone. The granules are usually closely packed but because of variations in size and spacing the ring has a broken outline. Stallard²⁸² compared the appearance of the granules to spots of white-lead paint. Although the central portions of these rings are usually clear, a few fine whitish dots may be scattered within their confines.

Examination in optic section discloses that the deposition in Bowman's zone results in an apparent depression. The overlying epithelium, which may be slightly thickened, shows no change in its surface contour. Vision is unaffected by the ring and no subjective symptoms are associated with it; the lesion is usually found accidentally.

There has been considerable speculation concerning the origin of these rings. Coats (who found the rings bilaterally) believed them to be congenital. Mayou²¹⁸ was of the same opinion. Ballantyne,¹⁰

longer, while in the lower half of the cornea the lines are shorter and much more numerous (Fig. 182).

In older individuals the double-contoured lines become thicker

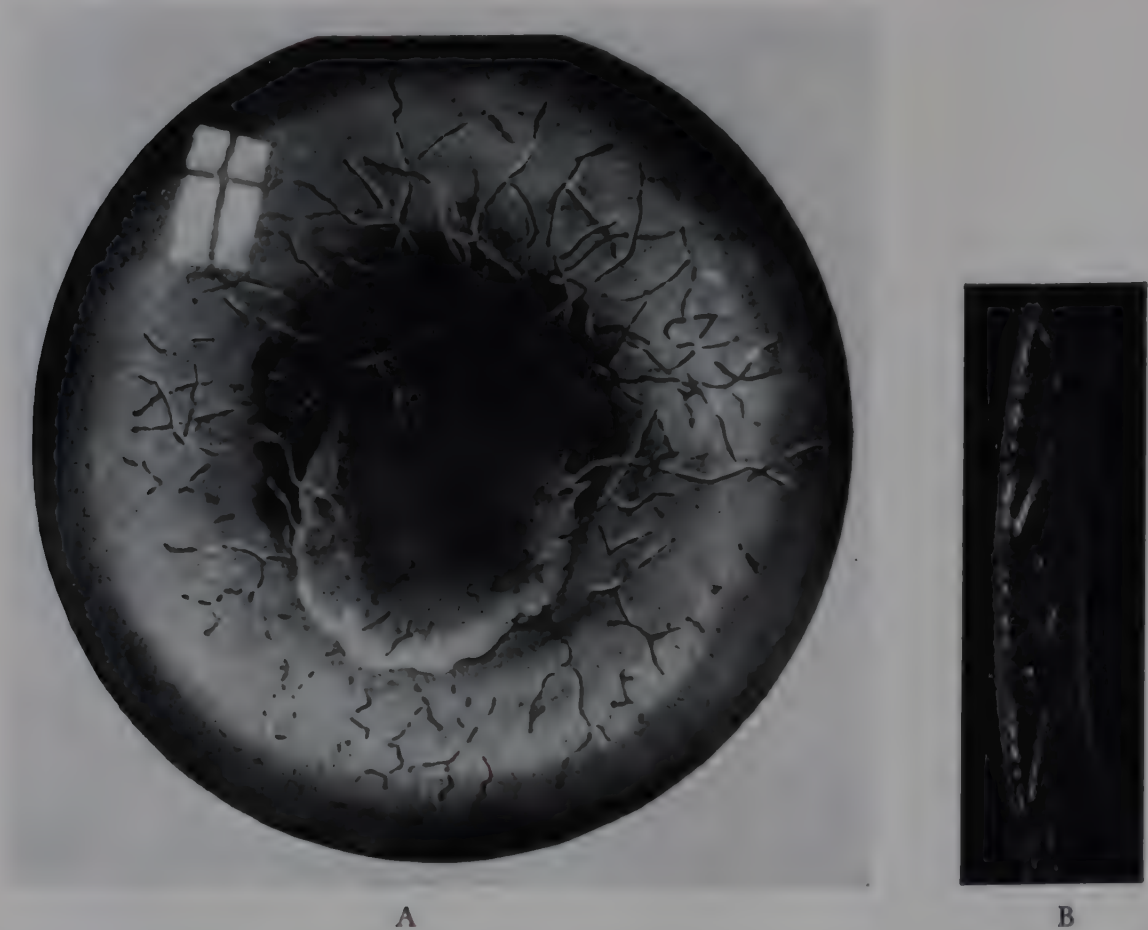


FIG. 182. Lattice-like dystrophy. A. Diffuse illumination. B. Optic section. (After Bücklers.)

and appear as optically empty channels. A disklike central opacity may also develop in the more advanced stages, which markedly reduces vision. Stainable surface erosions may occur occasionally, giving rise to photophobia, pain and congestion simulating inflammatory erosions. Owing to the irregularity of the surface, irregular corneal astigmatism results. Corneal sensitivity may be markedly reduced.

In biomicroscopic examination of young persons, fine lines are sometimes seen which may be confused with nerve fibers and may even appear as though connected with them. This has caused some writers to believe that the condition is due to a trophic disturbance; but vital staining (brilliant cresyl blue) has shown that the lines or tubes and the nerve fibers are not actually connected.

In diffuse illumination, the larger and more superficial figures appear as whitish, curved, poorly defined stripes on a gray background. By retro-illumination these lines (which may be double) appear brown, when viewed in front of the pupillary glow produced by trans-scleral illumination (Trantas). At the junctions of the intersecting lines small glasslike nodes or star-shaped maculae are seen. These nodular excrescences may raise the epithelium, which in turn may become eroded.

With the broad beam, in parallelepiped, a large number of small irregularly scattered short striae are seen between the coarser lines. Ultimately, this leads to a loss of corneal transparency. The lines or tubes have rather sharp edges which appear finely serrated by high magnification. The extremities of the tubes are tapered and lose themselves in the periphery but never quite reach the limbus. In later stages, a disciform or arclike opacity develops in the central portions of the cornea and is demonstrable by sclerotic scatter. This concentrically situated cloudy opacity is found in the sub-epithelial layers and may involve the entire corneal thickness.

Study with the optic section shows that the lines comprising the reticular network may run parallel to the surface or obliquely through the corneal thickness. They appear as optically empty tubes, which may be linear, fusiform, ovular, or round in shape, according to the direction in which they run and the angle at which the section cuts them. If the narrow light beam illuminates these fibers along their length, an oblique course in an anterior-posterior direction is found, while if the beam cuts the peripheral portions of the line at right angles only small holes are seen.

Haab¹⁴² considered that these lines were due to tears of the parenchyma or Bowman's membrane filled with a clear hyaline substance. Stanka²⁸⁴ and Greenwood¹³⁴ believed the structures to be water-clefts. According to more recent opinion this disease is due essentially to an interlamellar hyaline deposition causing subsequent disintegration of the corneal substance, and destruction of Bowman's membrane.

the onset has been observed between the ages of 9 to 13 years. As a rule the changes are unobservable with the unaided eye until the second decade (Fig. 180).



FIG. 180. Reticular dystrophy showing tubular structures in the parenchyma and annular opacity in Bowman's zone. In parallelepiped the tubular structures appear solid; retro-illumination reveals their tubular nature. (After Lloyd.)

With the development of the lesion a system of interlacing lines and tubes in the superficial corneal stroma is seen with a matt appearance of the cornea simulating thin Japanese lens tissue. In the early stages the parenchyma surrounding these structures is fairly clear, but later, as the reticular lines and tubes increase, it becomes more relucet. As the pathologic picture develops the changes consist more and more of these lines, which very often extend in radiating directions, but they may also extend concentrically with the center of the cornea. The branching interlaced reticular structures appear symmetric and may extend in all directions from the center or may be confined more or less to one area in some instances (Fig. 181).

Bizarre designs formed by the interlacing lines have been described which have been given fanciful names, such as alphabet type. Linear opacities that may develop in the nodular type of

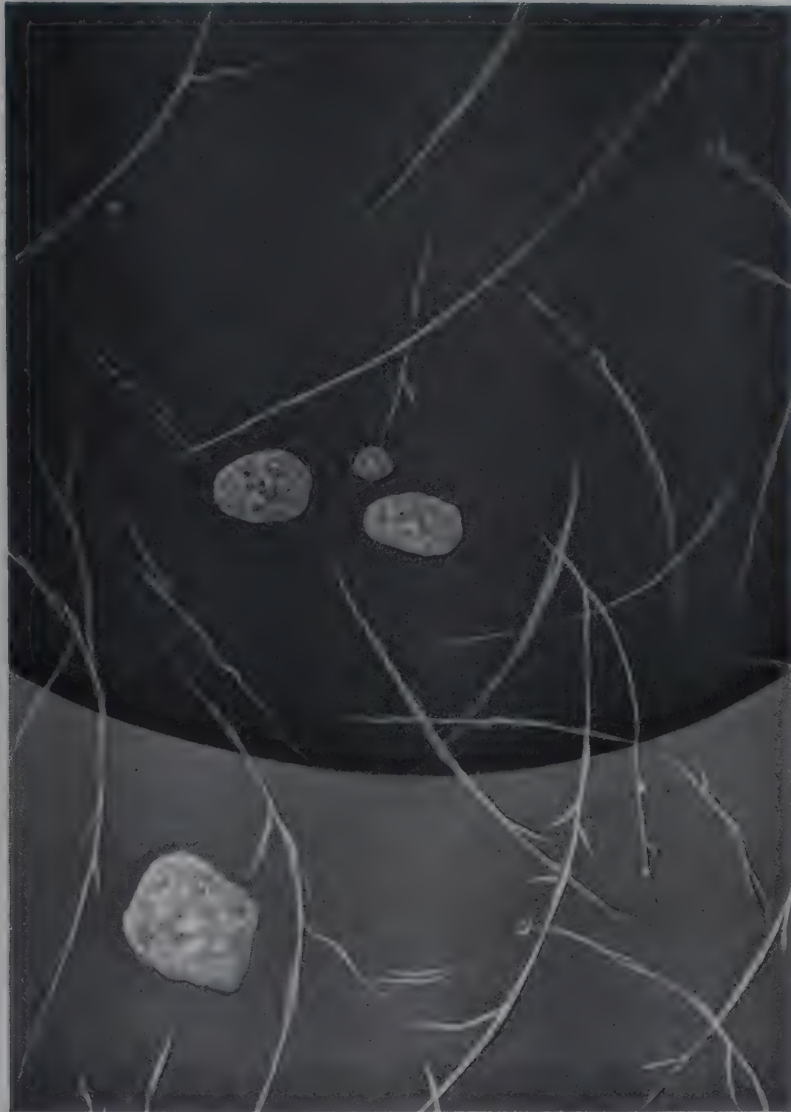


FIG. 181. *Dystrophia corneae reticulata*. Lattice-like dystrophy. (After Lloyd.)

corneal dystrophy have been incorrectly interpreted as lattice-like formations. Bücklers⁴⁰ stated that it is impossible to find combined forms, showing mixtures of nodular, macular and reticular types, and that, genetically, each type is transmitted in pure form; a parent with the nodular type will transmit only this form to his offspring. He believes that only those cases in which the opacities eventually appear as double-contoured lines should be considered as true reticular corneal dystrophy.

Bücklers observed in his cases that the lines may be concentrated more in the upper half of the cornea where they are thicker and

Like the nodular type this dystrophy begins in the first decade. The first signs are seen in children between five and nine years of age. At this age biomicroscopic examination of the corneae reveals tiny white opacities, consisting of very fine, not quite sharply circumscribed, subepithelial spots of varied form. By sclerotic scatter, the entire cornea appears as though covered by a veil, owing largely to increased relucency in the region of Bowman's zone. In addition, the deeper parenchyma may already contain several small spots.

Between ten and twelve years of age the opacities increase rapidly in thickness and in extent so that the children themselves notice a diminution of visual acuity. This early diminution in vision is caused not only by these poorly defined spots but also by a diffuse parenchymal haze to which unevenness of the epithelial surface is soon added. In consequence of the further development of the subepithelial deposits, a roughening of the corneal surface results; this assumes a dull dappled character but never becomes as involved and raised as in the dominant nodular dystrophy. Toward the periphery, rounder gray opacities may be found in the deeper anterior parenchymal layers.

Beginning with the third decade one must distinguish between a fine spotty and a coarse spotty type, both of which may appear concurrently in a familial group. The boundaries between these various spots are poorly defined. At first glance, they remind one of infiltrates or precipitates but no inflammation or neovascularization is ever seen. Still later the discrete foci tend to become more compact by continued coalescence of these small white dots in the anterior parenchyma or they may become pushed together to form a discoid figure. At times the macular opacities have threadlike projections, extending into the deeper layers of the parenchyma. The visual loss is progressive and in many instances may be almost total.

In the fourth decade, larger plaques are recognizable with the unaided eye. Very often these plaques join to form rings and cover the area of a moderately dilated pupil. The epithelium remains intact in every case. Often, early in the course of the disease, intensely

brown pigment lines (Stähli-Hudson lines) develop, running horizontally across the middle of the cornea. These may have forked extremities or they may join axially to form small spirals.

Corneal sensitivity may or may not be diminished. Spontaneous inflammation of a moderate degree is noticed in many cases, as evinced by photophobia and vascularization. This may disappear spontaneously without treatment. During the attacks of spontaneous inflammation there may be an increase in the opacities, which resemble inflammatory infiltrations.

With the biomicroscope, sclerotic scatter reveals a parenchymal haze pervading the entire cornea, with areas showing denser scattering in the region of the more opaque maculae. By proximal illumination the spots are seen more distinctly and a large number of smaller opacities are discerned between the larger opacities. More deeply seated, rounded and flattened opacities are especially visible at the periphery. By retro-illumination the larger spots are resolved into a somewhat crumbly appearance and do not have sharp outlines. In the broad beam, opacities in the parenchyma appear amorphous, especially at the periphery. Optic section reveals variations in corneal thickness brought about through elevations or depressions of the corneal surface. The elevations are situated over large dense infiltrations in Bowman's zone.

The maculas, seen in optic section as short, vertically placed whitish gray streaks or rods in the parenchyma, give an irregularly laminated appearance to the section in the pupillary region. In some of the advanced cases the width of the section becomes markedly diminished in these areas. A clear view of the posterior face of the cornea in parallelepiped is usually unobtainable owing to the anterior opacities.

Lattice-like Type (Biber), (*Dystrophia Corneae Reticulata*, *Dominant Reticular Dystrophy* (Dimmer), *Grill-Like Keratitis* (Haab) (*Grillage*). The lattice-like or reticular form was first described by Biber²⁸ and then independently by Haab¹⁴² and by Dimmer.⁵⁷ Clinically, in this dominant familial corneal dystrophy

stroma was diffusely hazy. In these cases he pointed out that the haziness could extend to the limbus.

Specifically, with biomicroscopy, sclerotic scatter demonstrates a discoid opacity in the center of the cornea; the broad beam reveals (in parallelepiped) a marbled appearance of the corneal tissue, much like snowflakes against a gray sky. By retro-illumination, the deposits are crumblike and by proximal illumination an impression of glasslike or crystalline infiltrations is gained. In optic section, the smaller and more numerous granules are seen to lie in Bowman's zone while the coarser granules lie in the deeper layers of the parenchyma. The particles increase somewhat in the very aged but not to any great extent. No regular correspondence exists between the density of the deposits, the size of the opaque area, and the age of the patient with this exception: for all forms of nodular dystrophy the particles are sharply defined and lie embedded in clear corneal substance.

During youth the epithelial surface is smooth but in middle and old age coarser particles cause surface irregularities by elevating the epithelium. The corneal surface appears sandy or stippled in diffuse illumination. The epithelium is always intact and rarely stains with fluorescein.

The visual acuity correspondingly diminishes with the progress of the lesion. The corneal sensitivity seems to be diminished over the nodular infiltration, the clear areas being unaffected. As a rule the eyes are not inflamed and corneal vascularization does not occur. Goar,¹¹⁸ who made histologic studies in one case, reported progression leading to complete corneal opacification with considerable vascularization. Histologic study led to the conclusion that the nodular change was essentially of a hyaline nature.

Macular Type (Recessive Form of Groenouw's Familial Corneal Dystrophy Type II — Macular Corneal Dystrophy of Febr). The recessive nature of this dystrophy was established after the investigations of Febr and Bücklers. Consanguinity of the progenitors has been noted in nearly all instances, with the number of affected children increasing with the number of siblings (Figs. 178, 179).

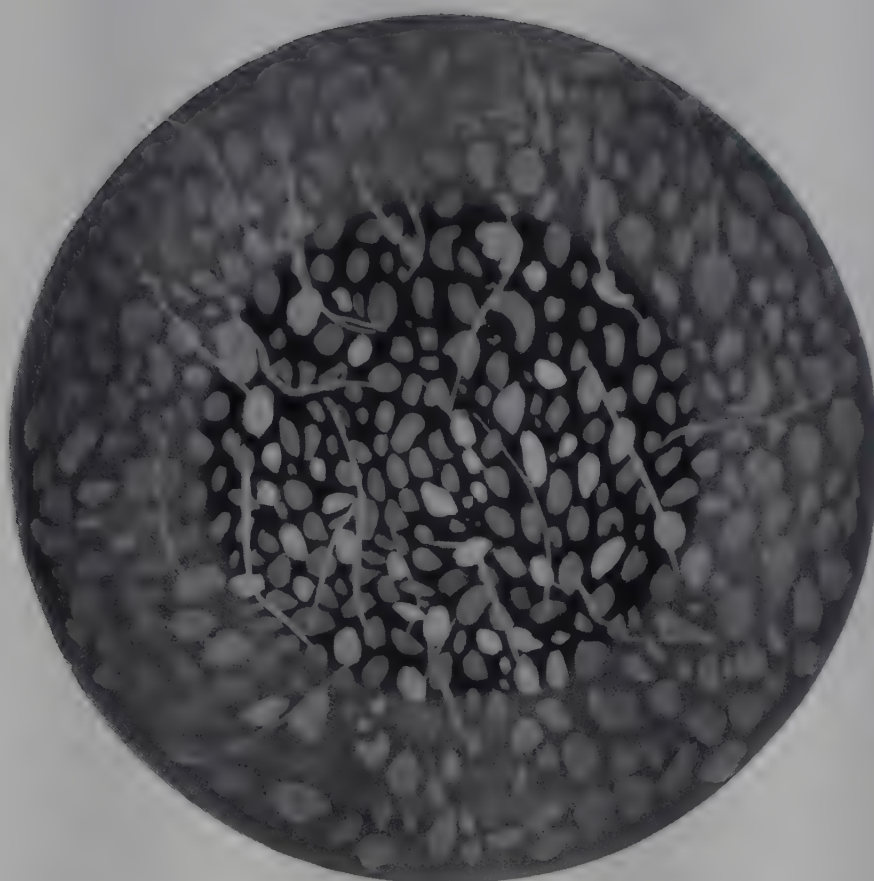


FIG. 178. *Dystrophia corneae maculosa*. (Franceschetti and Streiff. In *Modern Trends in Ophthalmology*. Ridley and Sorsby, Editors. Courtesy of Butterworth & Co.)

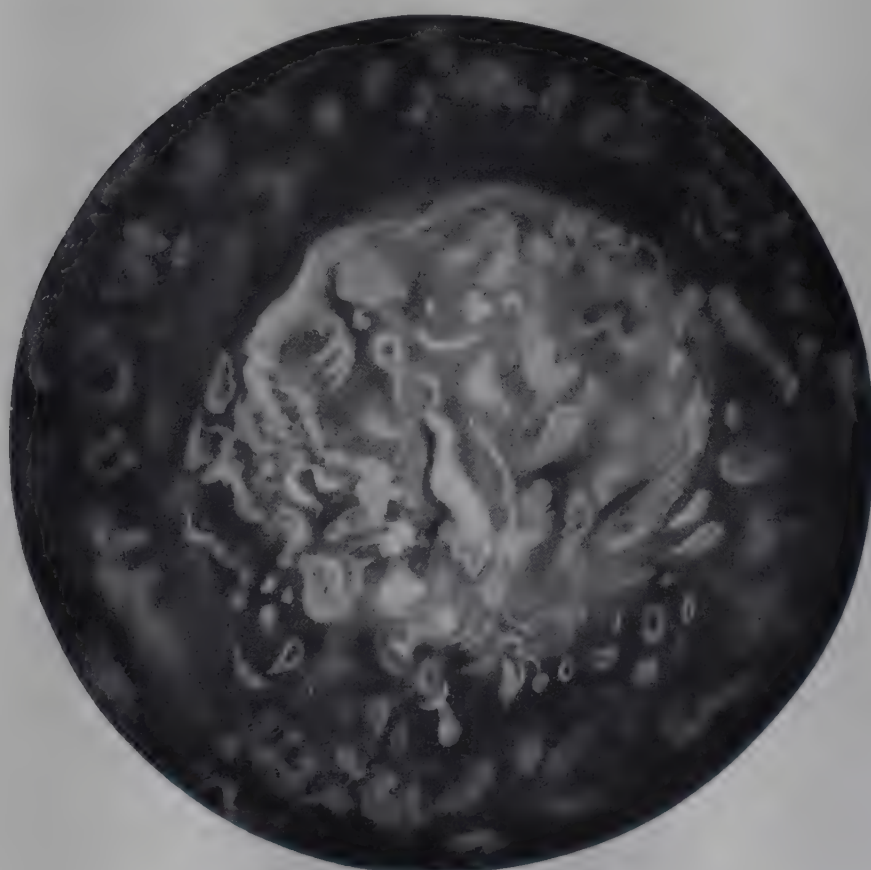


FIG. 179. *Dystrophia corneae maculosa*, advanced stage. (After Bücklers.)

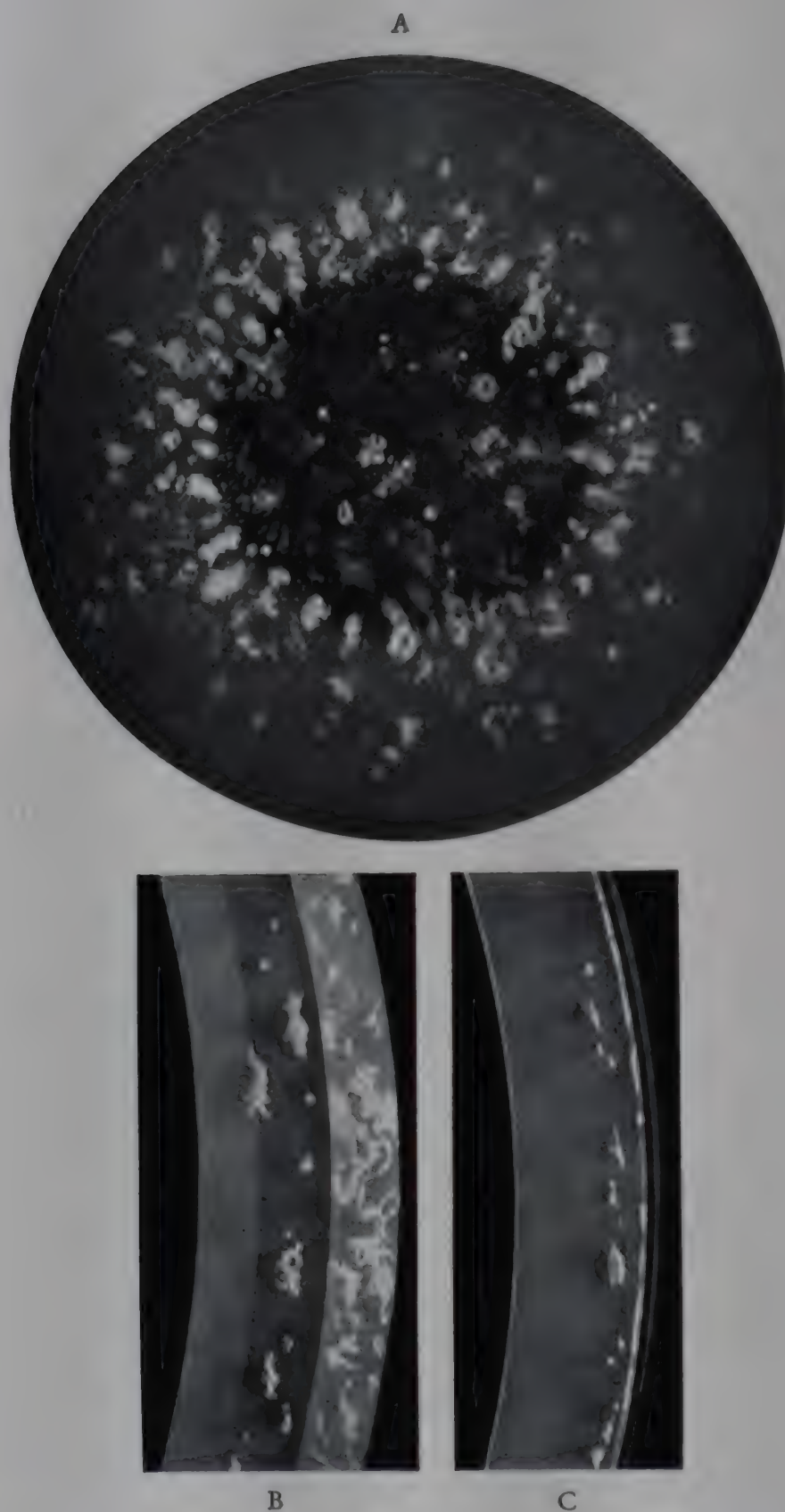
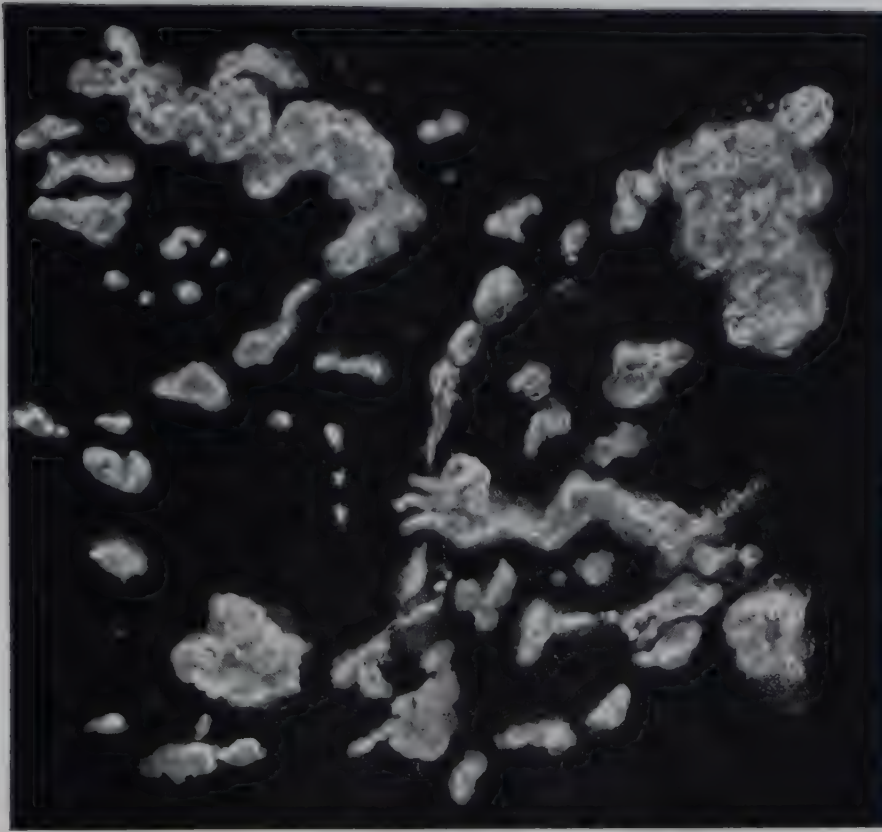


FIG. 176. Nodular dystrophy (Groenouw). Advanced stage. A. Disklike opacity almost involving the entire cornea, except for a clear zone near the limbus. B. Opacities seen in parallelepiped (direct focal illumination). C. Opacities in optic section. (After Vogt.)

A

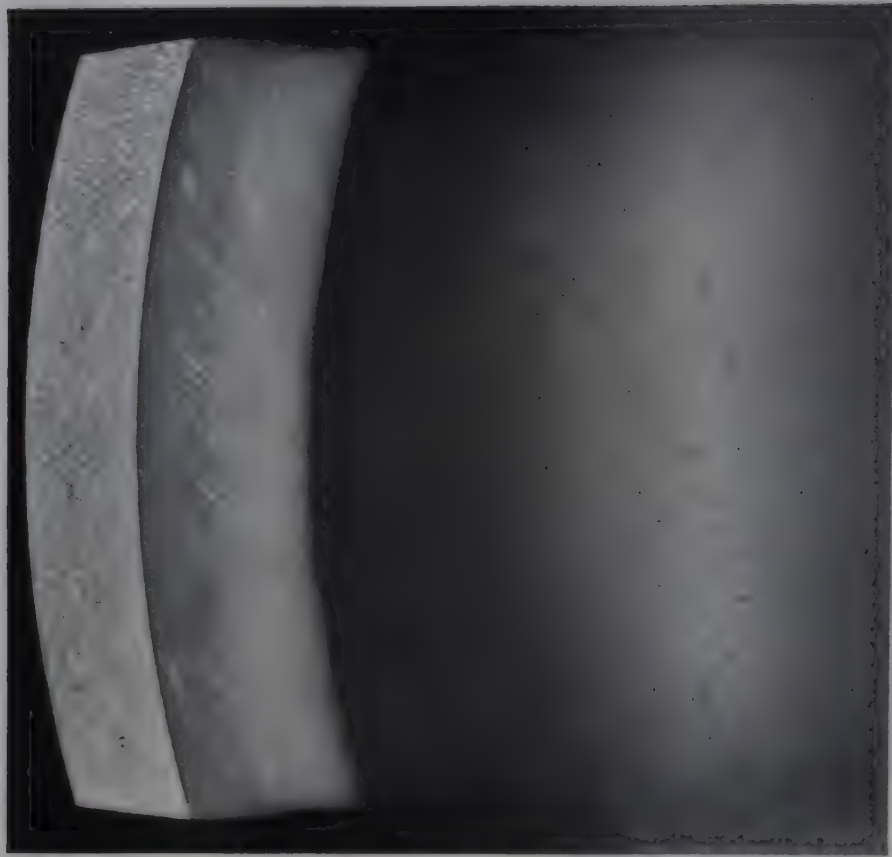


B



C

FIG. 177. A. Hyaline granules, irregular wormlike figures (darkfield illumination). B. Same structures seen in direct focal illumination parallelepiped. C. The smaller opacities appearing in the lower part of B, localized by optic section. (After Bücklers.)



A



B



C

FIG. 174. Familial granular dystrophy. A. Superficial punctate or granular lesions and changes in the stroma. (Direct focal illumination and retro-illumination.) B. Optic section. Granular lesion seen in Bowman's membrane composed of dots. Linear opacities in the parenchyma. C. Diagram illustrating the extent of the involvement by diffuse illumination.

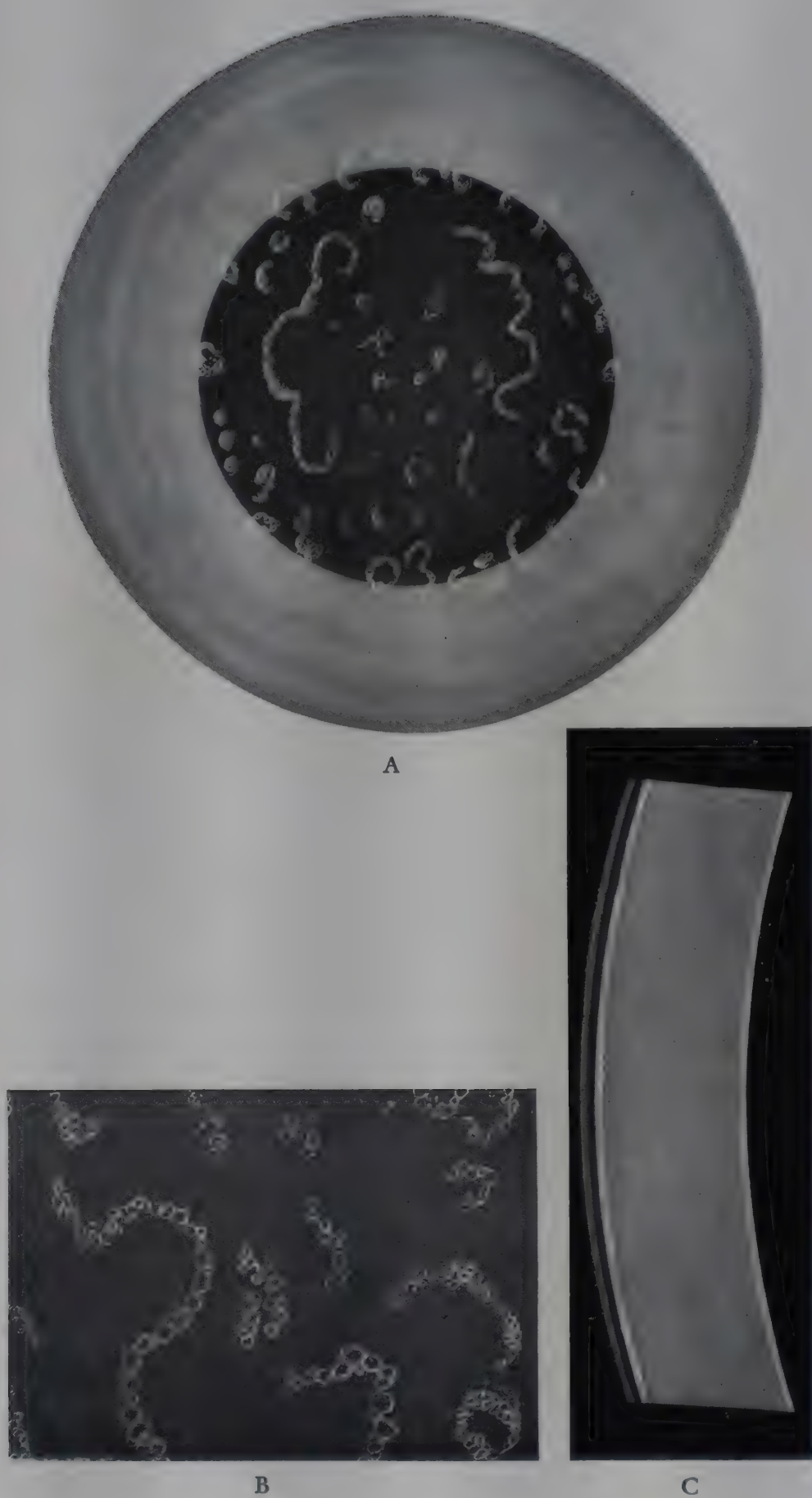


FIG. 175. Dystrophia corneae granulosa. Intermediate stages. A. Lesions in diffuse illumination. B. The same lesions in diffuse illumination under higher power. C. Optic section showing location of lesions in Bowman's zone. (After Lloyd.)



A



B

FIG. 173. *Dystrophia corneae nodulosa* and *granulosa*. A to D show four changes in the development of typical granular dystrophy. A. Small central punctate dots. B. Dots and radiating lines.



C



D

FIG. 173. c. Crumblike formations. D. Nodular aggregations arranged in the disk involving all the corneal layers. (Franceschetti and Streiff. In *Modern Trends in Ophthalmology*. Ridley and Sorsby, Editors. Courtesy of Butterworth & Co.)



FIG. 183. White rings of Coats showing location in cornea (left) and appearance by high power (right). (A and B, after Ballantyne; C, after Mayou.)

another, the appearance of a dystrophy may change with progression of the lesion.

Nodular Dystrophy. This form of familial corneal dystrophy begins in the first decade of life, usually becoming microscopically visible after five years of age as very small white dots, situated in the center of the cornea in the epithelial or subepithelial zones (Figs. 173). A characteristic appearance seen in the early stages is that of lines radiating from the center of the cornea or from an eccentric focus. These lines are thin and are composed of closely packed fine dots.

In some instances, whorl-like figures may form. In retro-illumination these dots appear as small droplets. It should be emphasized that these delicate early changes can be seen only with the biomicroscope; not until the dots or granules fuse to form large nodules do they become visible to the unaided eye (Fig. 174).

At the end of the second decade the pupillary region becomes more or less covered with small whitish figures, appearing as circles, arcs or disks, clubbed, pointed, crescentic, garlanded or sausage-shaped forms, but mostly as completely irregular forms. The larger nodules are established through conglomeration of smaller granular forms.

In the third and fourth decades, the opacity covers a discoid area involving the entire pupillary region or as much as two thirds of the corneal area. The concentration, size and distribution varies somewhat in different cases so that one eye bears no resemblance to another. Larger figures may be seen with the loupe but smaller ones can be seen only by biomicroscopic examination (Fig. 175).

In the fifth and sixth decades the region in front of the widened pupil is covered more or less diffusely with opacities. As the condition slowly progresses new spots appear deeper in the parenchyma but the peripheral cornea remains unaffected, leaving a clear circular band from 2 to 3 mm. wide between the opacity and the limbus (Figs. 176, 177).

Fleischer⁸⁸ described a ring-shaped variant of nodular dystrophy, in which the opacities were not well defined and the intervening

who reported six cases, suggested that the rings might be due to *Driisen* (wartlike) formation which had undergone calcareous degeneration. Gallemaerts¹¹³ and Vogt³²⁷ attribute the formation of



FIG. 184. Cornea farinata. Small punctate and linear deposits as they appear in the deeper parts of the parallelepiped and by retro-illumination.

these rings to some antecedent trauma. According to Duke-Elder,⁶⁷ "the most probable presumption is that it is an instance of secondary fatty infiltration of a small scar following a minute (and often forgotten) injury, such as that inflicted by a foreign body which had penetrated beneath the epithelium." This concept was strengthened by the fact that histochemical studies revealed that the opacity consisted of aggregations of lipoid material.²²⁸

Cornea Farinata. Cornea farinata was first described by Vogt³²⁴ in 1930. Since it frequently occurs in old people, this lesion has been considered to be an expression of senile physiologic loss of the transparent properties of the cornea (Fig. 184). Its resemblance to certain lipin dystrophies (i.e., lipin keratitis or Hurler's

syndrome) might suggest that it is a related senile lipoid disturbance.

Although cornea farinata is usually a bilateral affection of the deep layers of the stroma, it may occur in only one eye. According to Graves,¹³² it consists of a sprinkling of fine dustlike, respersive, colorless stipplings in the central pupillary portions of the cornea, confined to the deepest layers, and observed best in indirect retro-illumination. By direct retro-illumination, the fine refractile elements show unreversed illumination. In parallelepiped the deposit appears as fine white grouped relucant dots, forming an irregular mottling in the deeper parenchyma. These opacities are of minute dimension. High magnification is required for their resolution.

Superficial and Deep Crocodile Shagreen. Vogt cites an extremely rare degenerative condition of Bowman's membrane of obscure etiology, consisting of a central, thin, disklike opacity, the surface of which is traversed by dark streaks, probably caused by tears of Bowman's membrane and resembling crocodile shagreen (crocodile leather) (Plate XXVII, fig. 4). Deep crocodile shagreen, which was also described by Vogt, is similar to the former condition except that Descemet's membrane is affected in the axial region of the cornea. This latter (deep) type is apparently even rarer than the former.

Bandformed Opacity. The synonyms for bandformed opacity are: ribbon-shaped opacity, zonular opacity, calcareous film, and keratitis petrificans. This form of superficial corneal degeneration, which was first described by Dixon,⁵⁹ is characterized by the insidious evolution of a grayish bandlike opacity, situated in the exposed interpalpebral zone.

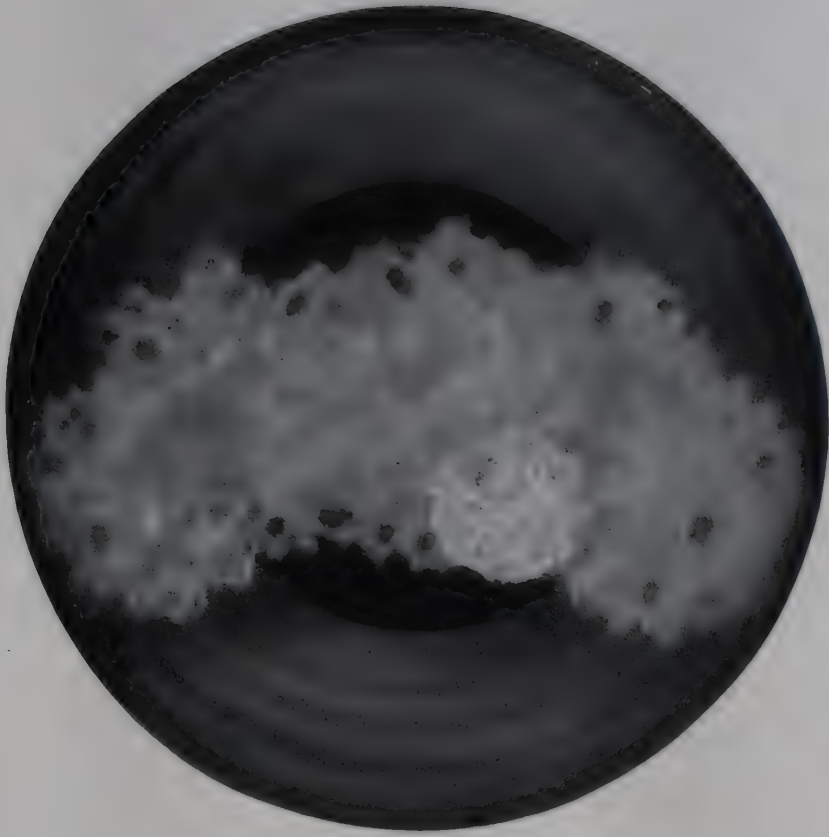
Three different etiologies are recognized as causing this type of degeneration: In one type, degeneration seems to begin spontaneously in otherwise unaffected eyes, and it is usually seen in the aged; but instances have been reported in the third or fourth decade. In another, the disease occurs in eyes which have been damaged by long standing intra-ocular infections. The lesion is usually found in degenerated and shrunken eyes, especially those affected with glaucoma following uveitis or associated with it. In rare instances, such a change may be seen following severe interstitial keratitis, and

in adolescents suffering from low-grade serous iritis, associated with rheumatoid conditions. Wong³³⁹ recently described a case in a boy, 13 years of age, associated with juvenile atrophic arthritis. Staining of a few of the opacities near the limbus was obtained two hours after an intravenous injection of congo red solution. Because of this Wong believes that the early changes are due to a deposition of amyloid or hyaline material. This finding is given added significance by the fact that amyloid deposits in the viscera and lymph nodes are a common feature of juvenile chronic arthritis. According to Sisson,²⁷⁴ deposits of phosphates and carbonates of lime and minute amounts of sulfur and silicon occur later.

The third or traumatic type occurs after exposure to irritants, such as mercury vapor,⁴⁷ calomel used as a dusting powder,¹¹¹ calcium bichromate vapor,²²³ naphthalene, and aniline dyes. Topolansky²⁹⁹ described this form of opacity in hatters whose eyes were irritated by small hairs freed during the processing of hare-skins.

Several forms of bandformed opacity may be seen. The most common variety is that in which a horizontal ribbon or band-shaped opacity, single or multiple, having dark round holes, extends across the cornea in the interpalpebral exposed zone (Plate XIX, figs. 6, 7; Fig. 185). Another form is seen at the limbus and may encircle the cornea or occur as an isolated semilunar superficial opacity (Plate XIX, figs. 4, 5). However, the peripheral type must be differentiated from white limbus girdle and the so-called annular degeneration in which the opacity tends to spread along the limbus, and not horizontally toward the center of the cornea. This latter form does not display the typical dark holes.

Characteristically, when viewed with the biomicroscope, the lesion begins as a turbid haziness in Bowman's zone. This may develop from either side of the cornea, spreading along a horizontal line to meet centrally within the exposed interpalpebral zone to form a band. The opacity has an appearance which is seen in no other condition; namely, small dark round fenestrations (holes) of varying size scattered throughout the opaque lesion. In contradistinction to the peripheral type, it is separated from the limbus by a clear zone (Plate XIX, fig. 6).



A



B

FIG. 185. Bandformed keratitis. A. Sclerotic scatter showing extent, location, and configuration of lesion. Small round dark areas represent the so-called holes in Bowman's membrane. B. Optic section showing the involvement in Bowman's zone as a white mottled opaque line (about 40 X).

The configuration and presence of the fenestrations which occur in this lesion are well demonstrated by sclerotic scatter. The opacity (under high magnification in optic section) is seen to be formed by a matrix of tiny grayish dots. As the opacity progresses, the older portions of the lesion become white and chalky, and may show minute rounded holes and larger transparent cracks. In these areas the changes may extend into the subjacent layers of the parenchyma. The increase in whiteness and chalkiness is probably due to hyalinization and calcareous changes. The dark fenestrations may be caused by the perforation of Bowman's membrane by corneal nerves. The clear fissures which appear later may be produced by cracks in the calcareous and degenerated Bowman's membrane.

The peripheral extremity of the lesion is usually sharply demarcated, while the progressive central borders are diffuse and blurred. After the band has fully formed, the upper and lower borders are hazy and may be serrated.

In the early stages, before extensive calcification has occurred, the film-line and epithelium may not be affected as they pass over the lesion. However, over the most involved areas epithelial edema may be seen by retro-illumination. As the condition progresses, dehiscences in the epithelium produce erosions. Small staining areas may appear. Such changes are evident, especially when calcareous incrustations begin to sequester. Unless ulceration with secondary infection occurs, there is no tendency toward corneal vascularization.

It should be noted that following the surgical removal of the calcareous incrustations, epithelization may restore varying degrees of transparency.

Lipin Keratitis. Several types of fatty corneal metamorphosis are grouped under this heading. The existing confusion regarding the nature of the changes involved in the so-called fatty degenerations and storages is reflected in the classification of their ocular manifestations. In the cornea it is usually considered as a primary or secondary change; the primary type is caused by some purely local disturbance of lipoid metabolism, or is associated with a systemic disturbance

of lipoid metabolism (e.g., Hurler's syndrome). It may also appear as a hereditary crystalline degeneration (Plate XIX, figs. 8, 9). In the so-called secondary forms, it is a late degenerative change in long-standing corneal disease. Whether or not such a classification is justifiable is still questionable, but in all probability the underlying pathogenetic processes are similar. The storage of lipins is intimately connected with the reticulo-endothelial system. Heath¹⁴⁷ states that "any mechanism precipitating or coarsening the normally fine fat emulsion can produce a foreign body type of reaction with great cellular response." (Plate XX, figs. 1, 2, 3, 4, 5, 6.)

Generally, the deposits which occur in the cornea are not neutral fat, but rather lipin in character. The chemical nature of the lipins seems to vary in different conditions. Experimentally, lipin storage in the eye has been produced by provoking hypercholesteremia in rabbits (Versé³¹⁸). In the degenerative type which follows long-standing diseases of the cornea, such as may arise from tuberculosis, syphilis, and repeated trauma the deposits may be irregularly distributed. In the primary type, the initial infiltration may start peripherally and extend centrally to form a discoid opacity. Later, its connection with the periphery may disappear. The lesions may clear spontaneously or remain localized and stationary, or they may progress to involve the entire cornea. Although the infiltration first appears in the deeper layers of the cornea, it may extend to the superficial layers and even cause epithelial changes (erosions).

In addition, the hereditary crystalline form must be considered as a lipin keratitis. This type is genetically dominant, and usually not included under the familial corneal dystrophies, as a distinct entity. A secondary form was first described by Schnyder²¹⁰; this consists of bilateral disciform opacity in the parenchyma, made up of closely packed short glistening needle-like crystals. Recently, I observed a case (through the courtesy of Dr. H. R. Sherman) in which there was a uniform deposition of varicolored crystals in Bowman's zone and the anterior parenchyma, entirely covering the corneae of both eyes (Plate XIX, figs. 8, 9). There was no loss of corneal transparency and visual acuity was unimpaired. The eyes

PLATE XX

FIG. 1. Lipin dystrophia. Diffuse illumination showing circumscribed area of infiltration in cornea; no vascularization present.

FIG. 2. Lipin dystrophia. Appearance of lesions (Fig. 1) in parallelepiped demonstrating the type and location of yellow punctate infiltrations.

FIG. 3. Hurler's lipin keratitis. Sclerotic scatter showing grayish cloud in the lower two thirds of the cornea.

FIG. 4. Details of Hurler's lipin keratitis (Fig. 3) in direct focal illumination. Appearance in parallelepiped showing small punctate infiltrations in the deeper cornea.

FIG. 5. Lipin keratitis. Central disciform type of lipin infiltration of ten years' duration, which appeared spontaneously in a woman, 40 years of age. Vascularization has appeared in the last two years. Diffuse illumination.

FIG. 6. Lipin keratitis. Optic section. Illustrating the location and character of deposit and the vascularization seen in Figure 5.



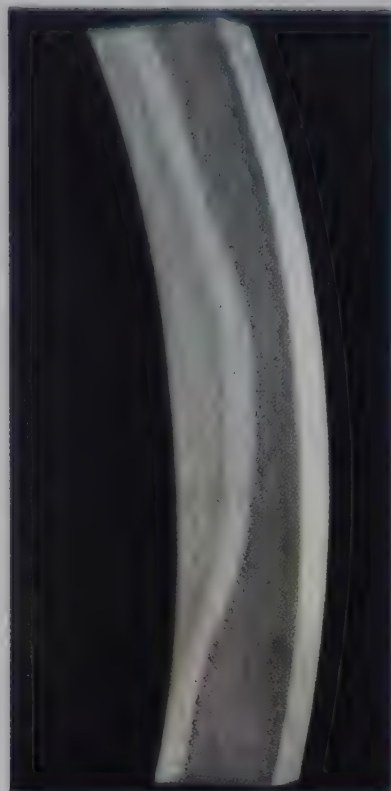
1



3



5



2



4



6

were otherwise normal, and there was not a history of irritation. No relevant family history was ascertainable.

I have reported three cases of lipin keratitis,²⁵ occurring in

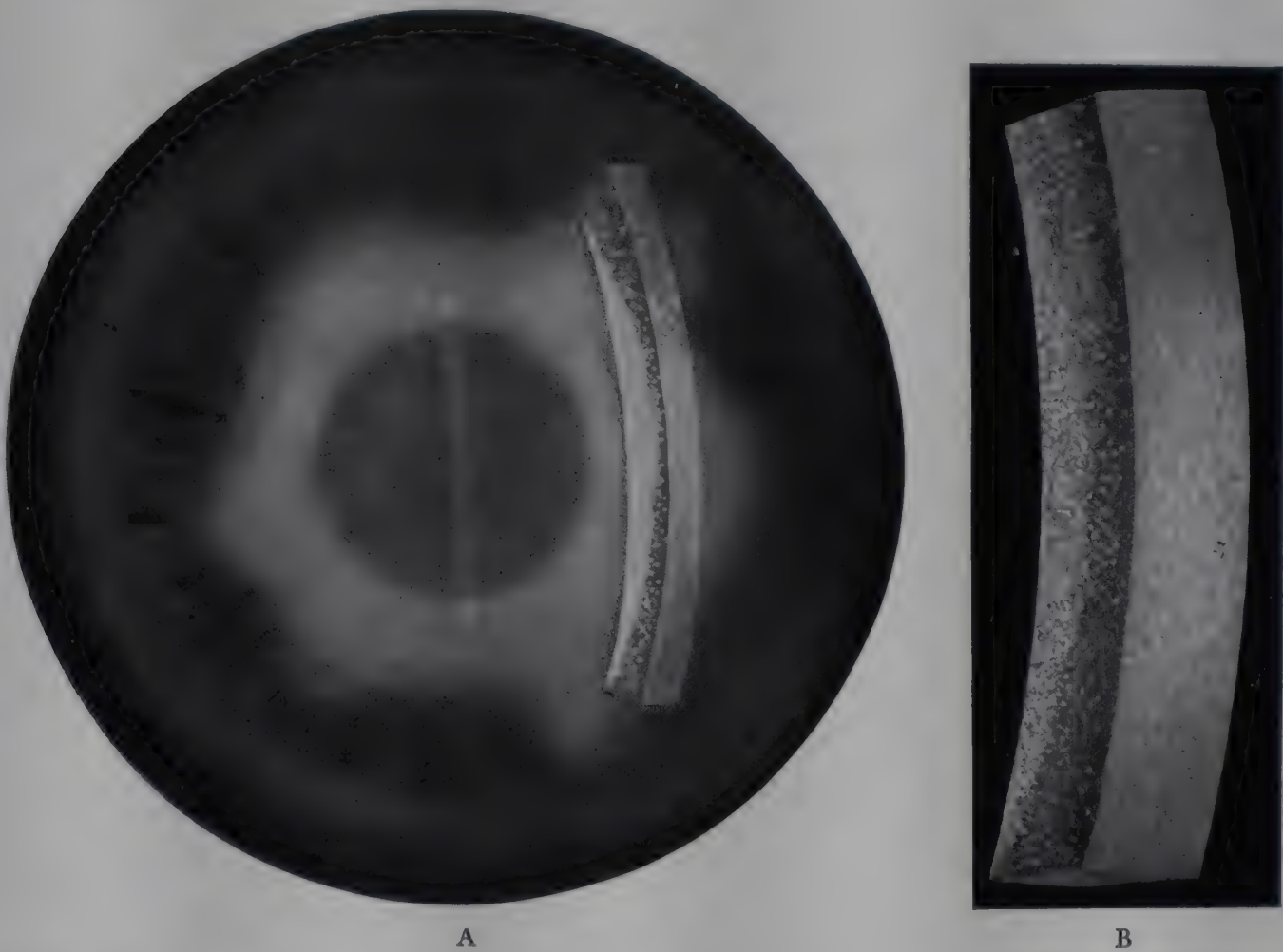


FIG. 186. Lipin keratitis in Hurler's syndrome. A. Early stage by direct and proximal illumination. B. Same stage by high power of parallelepiped.

Hurler's syndrome (gargoylism). One of these cases was examined by autopsy. This was the first time that a cornea in this condition had been examined biomicroscopically, and subsequently studied histologically (Plate XX, figs. 3, 4). With diffuse illumination, all three cases showed a characteristic milky haze, which at first occupied the pupillary areas, and then spread toward the periphery of the cornea. In optic section just below the epithelium, a few fine punctate dots were observed. The main infiltration, which began in the anterior middle layers and increased in density toward the posterior face, was seen as a collection of small punctate dots, varying in size (Fig. 186, A, B). A few fine brown pigment deposits were found on the deep face. No vascularization or edema was present. The char-

acter and disposition of the infiltration was similar in the three cases. The histologic examination of the cornea in one showed the presence of fine irregular lipin granules in interlamellar spindle-like spaces.

In an exhaustive review of the literature, Katz and Delaney¹⁶³ showed that a majority of the cases reported as primary should in reality be classified as secondary degenerations. In the cases reported by these authors (Katz and Delaney) the deposition began as a marginal infiltration, involving the entire periphery of the cornea, leaving the central areas of the cornea clear. A few years later, the infiltration progressed and both corneae became opaque. The opacity had a definite yellow hue and there was considerable peripheral, superficial vascularization. With the biomicroscope, the opacity seemed solid; the thin layer of less affected corneal stroma, which covered it, contained needle-shaped crystals. Histologically, interlamellar and intralamellar fat-laden histiocytes with scanty mitochondrial content, neutrophilic leukocytes, and swollen vacuolated corneal lamellae were seen.

Under the name *crystalline parenchymatous degeneration*, Vogt described a condition characterized by a patch of deep yellowish infiltration. At the borders of such lesions, he found a more or less clear narrow interval, which he called "lucid interval"; this clear area separates the main lesion from a surrounding fringe of needle-like crystals. The condition occurs in persons of middle or advanced age, and usually progresses slowly. Its etiology is unknown and there is an absence of inflammatory signs. I have seen similar cases, which I classified as primary lipin keratitis, unassociated with systemic disease or even with hypercholesteremia. The deep yellowish color seen in some of these infiltrations may be caused by lipochrome pigment. In one of my cases (Plate XX, figs. 5, 6) deep vascularization and renewed storage appeared several years after the initial infiltration.

Parker Heath believes that crystalline parenchymatous keratitis (Vogt), keratitis centralis, keratitis profunda, the misnamed fat "dystrophy" of the cornea, and xanthomatosis of the cornea — considered by many as separate entities — should all be grouped as lipin interstitial keratitis. He divided the progress of these chronic

lesions into three stages: (1) early, active lesion, (2) mature lesion, and (3) retrograde lesion.

The *early stage* is initiated by an infiltration of fine droplets in the parenchyma anterior to Descemet's membrane; this is associated with a loss of transparency. Vascularization may be absent but usually deep vessels penetrate the parenchyma early and are accompanied by a cloudy haze surrounding the areas of denser storage. Such lesions, if they are small, may clear up entirely; but if they are progressive they enter the second stage or stage of maturity.

The *mature lesion* is generally marked by more infiltration, causing an increase in corneal thickness, which at times results in the formation of a typical posterior corneal bulging (commonly seen in interstitial keratitis and in disciform keratitis), surrounded by a secondary concentric "reflection" ring and by an increase in the number of penetrating vessels. Iritic irritation may also occur. The older original infiltrations assume a yellowish tinge. The fresher infiltrates invade the more anterior parts of the parenchyma until the whole corneal thickness may be occupied by a dense opacity, comprising two thirds of its total area. With high power, the infiltration may be seen to be flocculent and partly amorphous, containing many fine admixed crystals. The crystals may occupy a fringelike zone separated from the main body of infiltration. The superficial parenchymal areas near the limbus may be clear, as in arcus senilis, but the deeper peripheral areas reveal the paths of invasion.

The *retrograde lesion* is characterized by lessening of storage, attenuation of vessels, clearing in some areas and scarring in others. Folds in Descemet's membrane and anterior concentric arclike rings of varying density appear. The remaining, permanent opacities usually show the presence of crystals. Heath reported that the prognosis was better in younger patients.

KERATOCONUS (CONICAL CORNEA, STAPHYLOMA PELLUCIDUM CONICUM, ANTERIOR MYOPIA)

Biomicroscopic examination has contributed much toward the understanding of the changes occurring in this obscure condition. It has been described as a "hyperbolic ectasia of the cornea of non-

inflammatory origin." Since keratoconus usually appears during adolescence, it has been regarded by ophthalmologists as the result of an aberration in development (possibly hereditary),* and by others as secondary to some general disturbance, resulting either from an endocrine dyscrasia or from avitaminosis. In some ways this condition resembles myopia. Both are characterized by tissue elongation. In a recent article by Terry and Chisholm²²⁵ on keratoconus, in which they described their successful experience in applying pressure to cure the corneal deformity, it was concluded that, "thinness in the central area of the cornea appears in the embryo and persists through life, representing a physiological keratoconus. The tensile strength of the cornea depends primarily on the white fibers of the substantia propria and the forces binding them together. Secondarily, elastic fibers lend strength when the cornea is distended. If elastic tissue is an important constituent of the cornea, conditions causing elastic-tissue degeneration, such as stria gravidarium, pseudo-xanthoma elasticum, pinguecular formation . . . may be of some etiological importance in keratoconus. As no lamellae extend over the entire cornea, any overdistention sufficient to disrupt the connection and adhesion of the lamellae would cause a slipping of the layers, thus producing an ectasia of the cornea. The greater number of lamellae at the periphery strengthens the cornea there."

Keratoconus seems to occur more commonly in females. Although it is bilateral, it may appear in one eye long before the second becomes involved. The condition tends to run a long chronic course, at times becoming arrested; when it is progressive it results in an apical scar. Acute ectasia results when rupture of Descemet's membrane per se occurs, leading to imbibition of aqueous fluid, with consequent swelling and opacification of the cornea. Regeneration of the endothelium causes resolution of the process by closure of the ruptures in Descemet's membrane and gradual clearing of the cornea. Complete rupture of the thinned cornea has occurred (although more frequently dense scarring prohibits this accident).

* Sanders²⁶⁵ reported a family (father and three daughters) affected with keratoconus and anterior polar cataract.

When viewed in diffuse illumination, the cone, which generally lies a little below the center of the cornea, presents a characteristic appearance, caused by irregular reflection; which has been compared to the luster from a "dew drop" or a "drop of water on a glass surface." This picture tends to disappear with the development of scarring.

Pathologic investigations have substantiated the more recent bio-microscopic findings of Vogt and Koeppe. Von der Heydt³²⁸ and Appelbaum² classified the corneal changes into seven distinct types of alterations, which may appear at varying periods during the course of the disease, although all of these may not be found in every case. These changes are as follows:

(1) *Thinning of the cornea at the apex of the cone.* This important change is practically always present, and can best be demonstrated by examination with optic section. The thinning may become so marked that the anterior and posterior limits of the optic section may seem almost in contact with one another (Plate XXI, fig. 3). Actually the thickness of the cornea at the summit of the cone may be reduced to one third that of the periphery. At times, because of the excessive corneal curvature, it may be difficult to keep the entire section in exact focus at one time.

(2) *Reflex from the endothelial cup.* This brilliant reflex, which is seen at an early stage before the parenchyma becomes opaque, accounts for the characteristic "dew drop" or "piece of crystal" appearance. The increased cuplike concavity of the posterior corneal surface of the apex intensifies its reflective properties, acting like a convex mirror.

(3) *Striae.* A series of short single whitish vertical or oblique lines, more or less parallel, are usually seen in the middle layers of the cornea (Plate XXI, fig. 1; Fig. 187). They probably are tension lines caused by stretching of the corneal lamellae and are almost pathognomonic of keratoconus. These striae are seen in the region of the apex before it becomes densely scarred, although in one case I have seen, they were situated on the slope of the conical cornea near the base of the cone. Frequently, observation by high power reveals

PLATE XXI

FIG. 1. Keratoconus. Diffuse illumination. Apical scar and striae in stroma; sickle-shaped sector of Fleischer's pigmented ring is seen at the right, separated from the main lesion by a clear area.

FIG. 2. Early stage of keratoconus (distal to apex of cornea) showing increased visibility of corneal nerves and an arc of Fleischer's ring. Diffuse illumination.

FIG. 3. Keratoconus under contact glass. In direct focal illumination (to the right) surface of the contact glass is stained with fluorescein. Scars and striae seen in stroma (to the left) which is thinned at the apex of the conus.

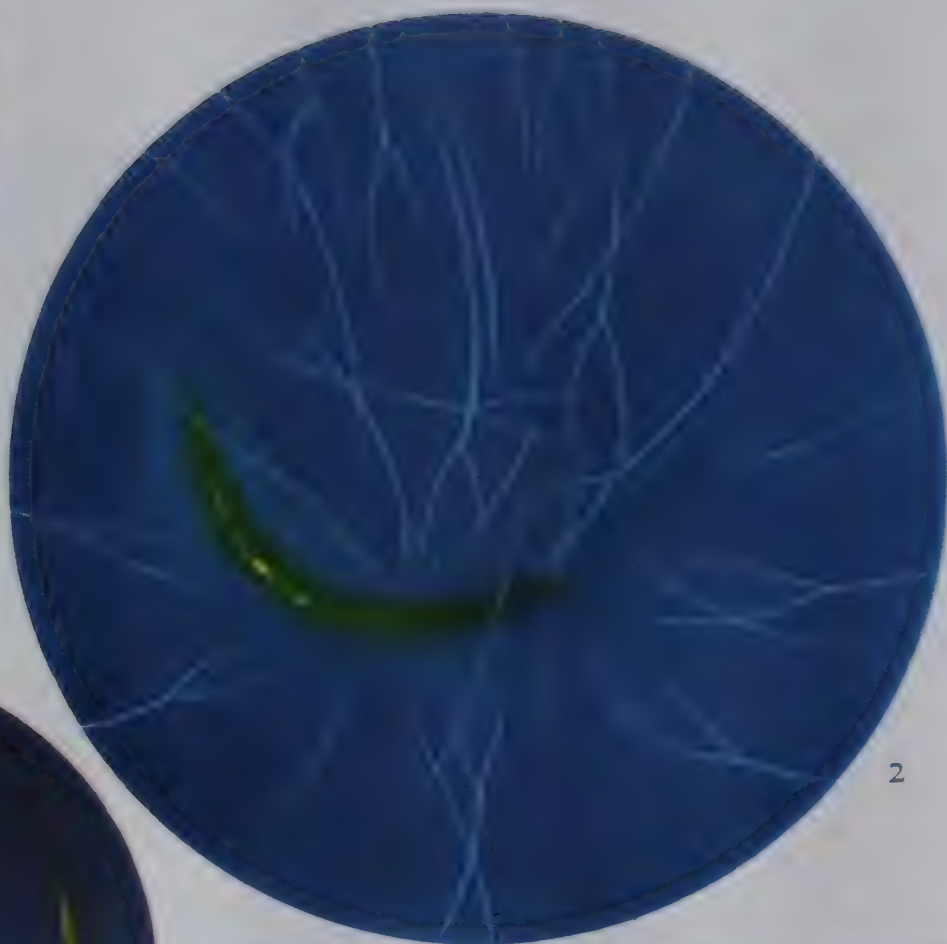
FIG. 4. Marginal dystrophy. Crossed folds in Descemet's membrane (pupillary area) and peculiar pigmented ring in Bowman's zone below. Direct focal and retro-illumination.

FIG. 5. Folds in Descemet's membrane.

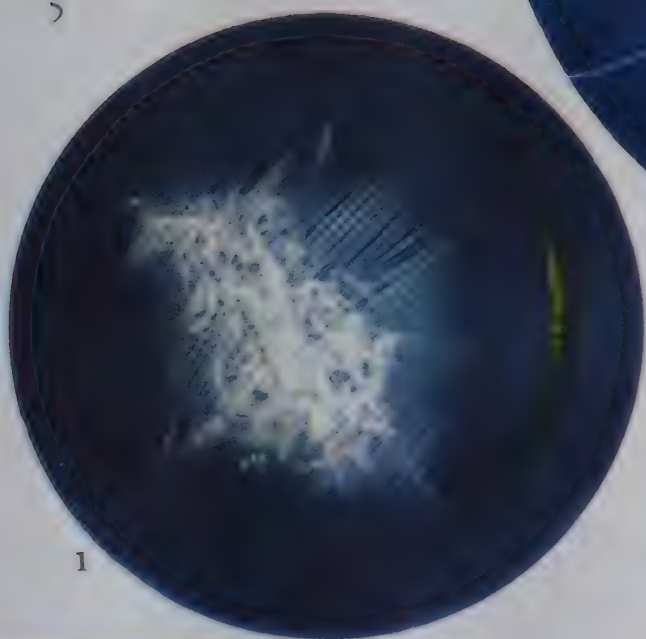
FIG. 6. Detailed view of the end of a fold in Descemet's membrane. Retro-illumination. 40 X.



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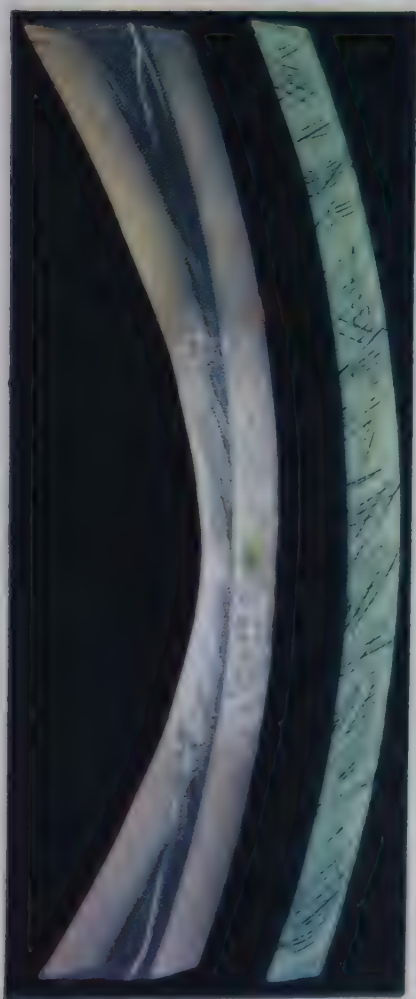
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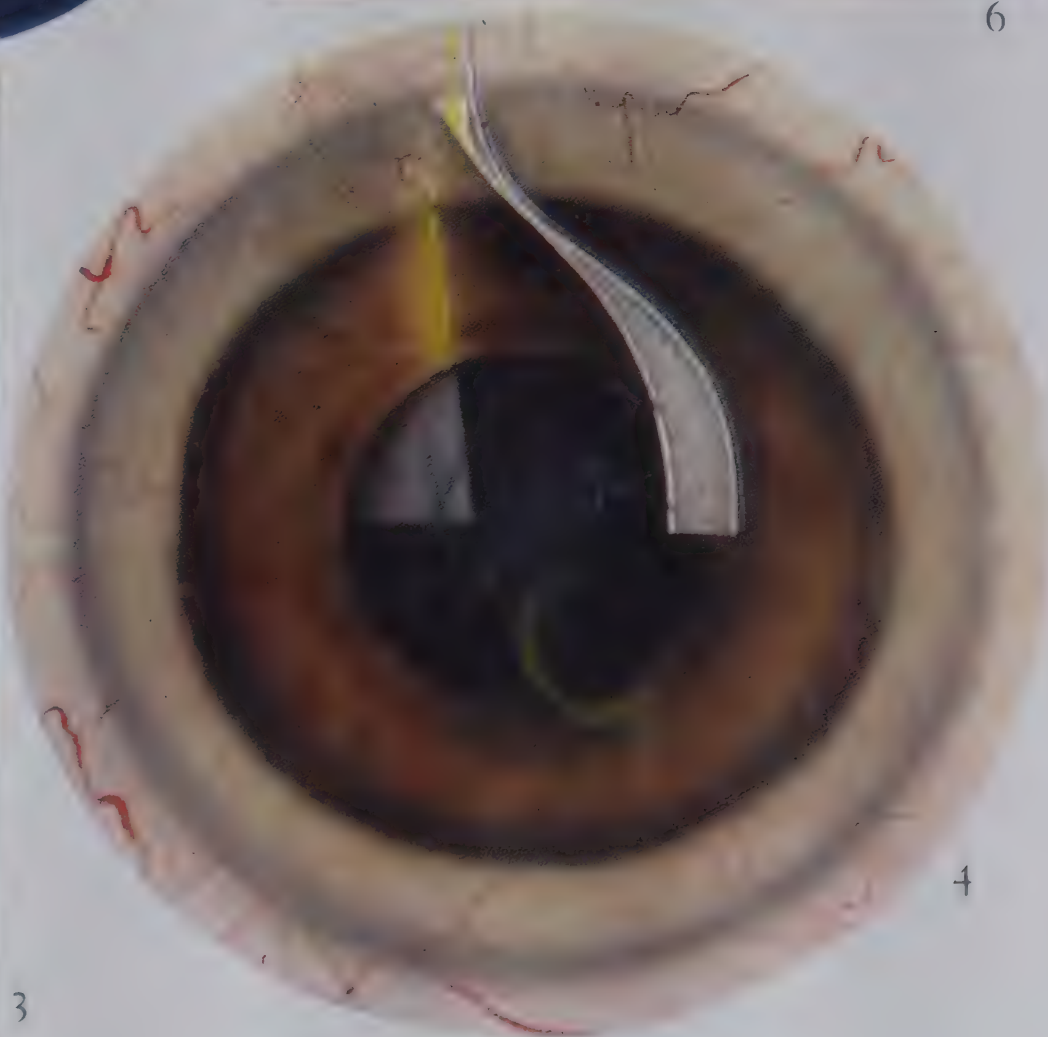
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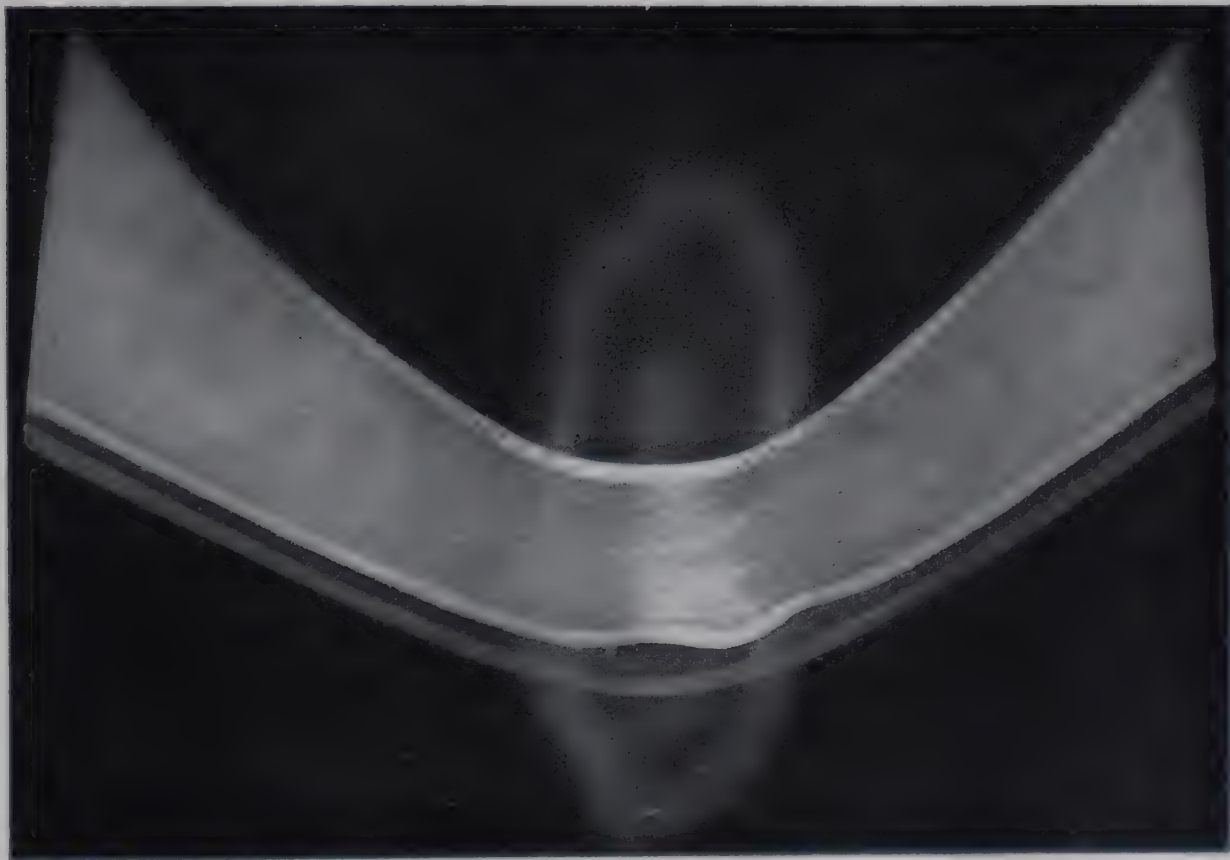


FIG. 187

FIG. 187. Keratoconus in optic section. Typical configuration of section. Apex of cone reveals folds in Bowman's membrane, striate, thinning and scarring of the parenchyma and endothelial changes.

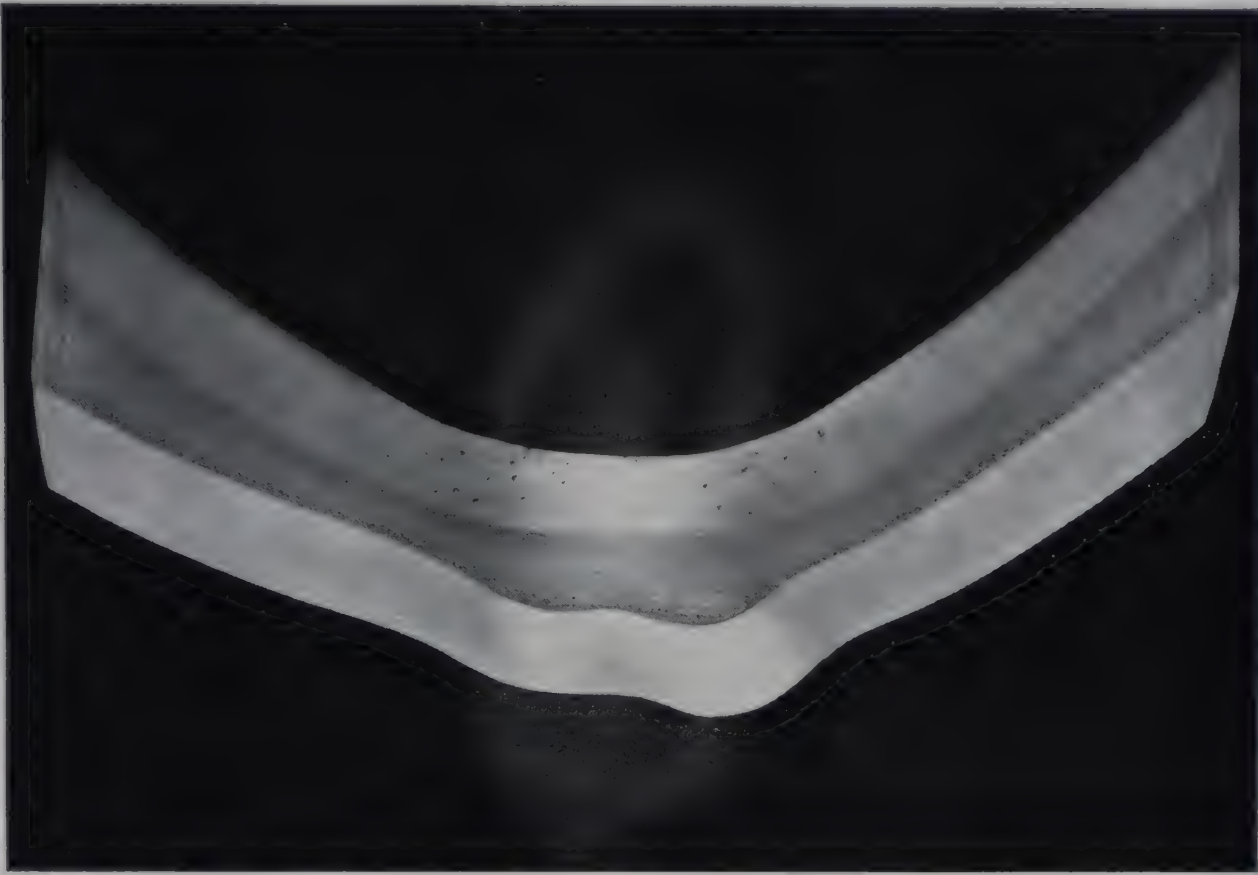


FIG. 188

FIG. 188. Keratoconus. Direct focal illumination with wide beam. Appearance of the apex showing opacities and folds in Bowman's membrane; changes in the substantia propria and posterior corneal surface.

delicate anastomosis of these lines. Crossing systems of striae may produce a lattice-work design. As a rule the lines do not cross at the same level. These striae were first seen by Elschnig in 1894, with a

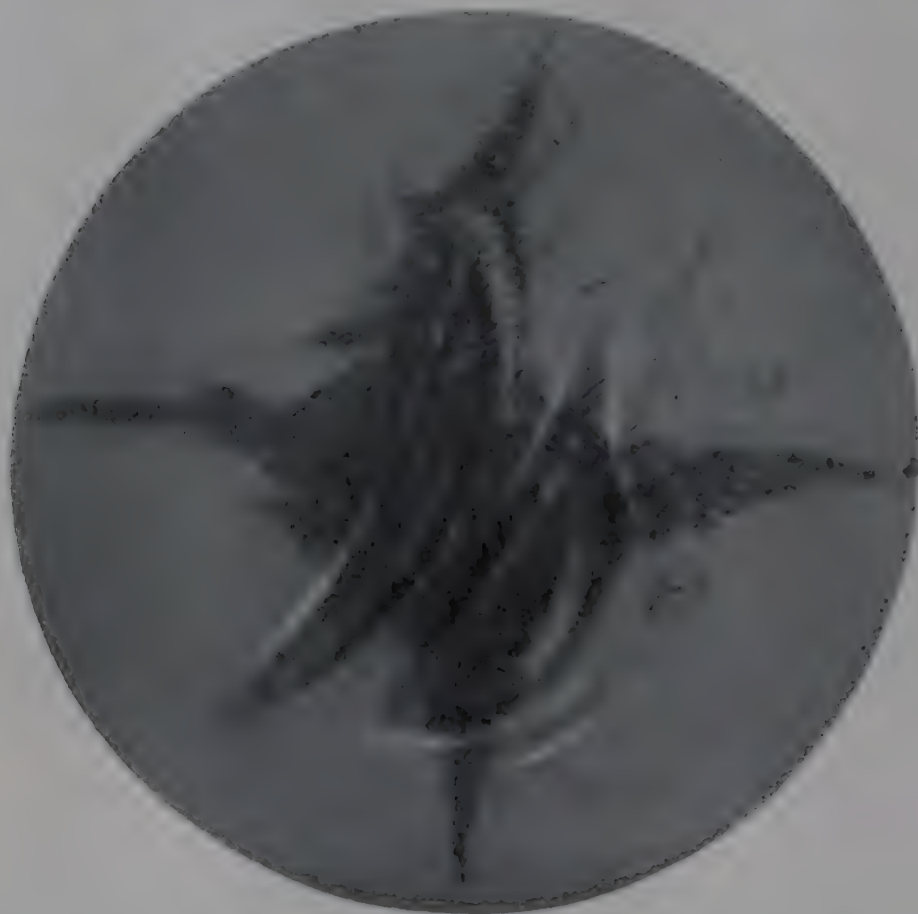


FIG. 189. Details (ruptures and scars) at the apex of the cornea as revealed by retro-illumination.

loupe, but later were thoroughly demonstrated by Vogt with the biomicroscope. Rados²¹⁵ attributed them to opacification of the elastic fibers (lamina elastica corneae). They differ in many ways from the superficial opacities and scars which are situated more anteriorly.

(4) *Irregular superficial corneal opacities or scars.* These opacities form on the apex of the cornea in the advanced stages and account for a considerable loss of visual acuity (Fig. 188). They commence as grayish dots, linear or confluent, and in optic section they are localized in Bowman's zone. Later, the spaces between the lines or groups of opacities become opaque and an irregular superficial opacity forms. These changes are caused by ruptures in Bowman's membrane, which are followed by filling of the rents with fibrillar connective tissue (Fig. 189).

(5) *Ruptures in Descemet's membrane.* This is not a constant occurrence in keratoconus, but when present it is characteristic.

The appearance varies with the size and extent of the rupture. Small ruptures may be semilunar or crescentic in shape, while larger ones may be sinuous. The rolled edges of a tear may cause

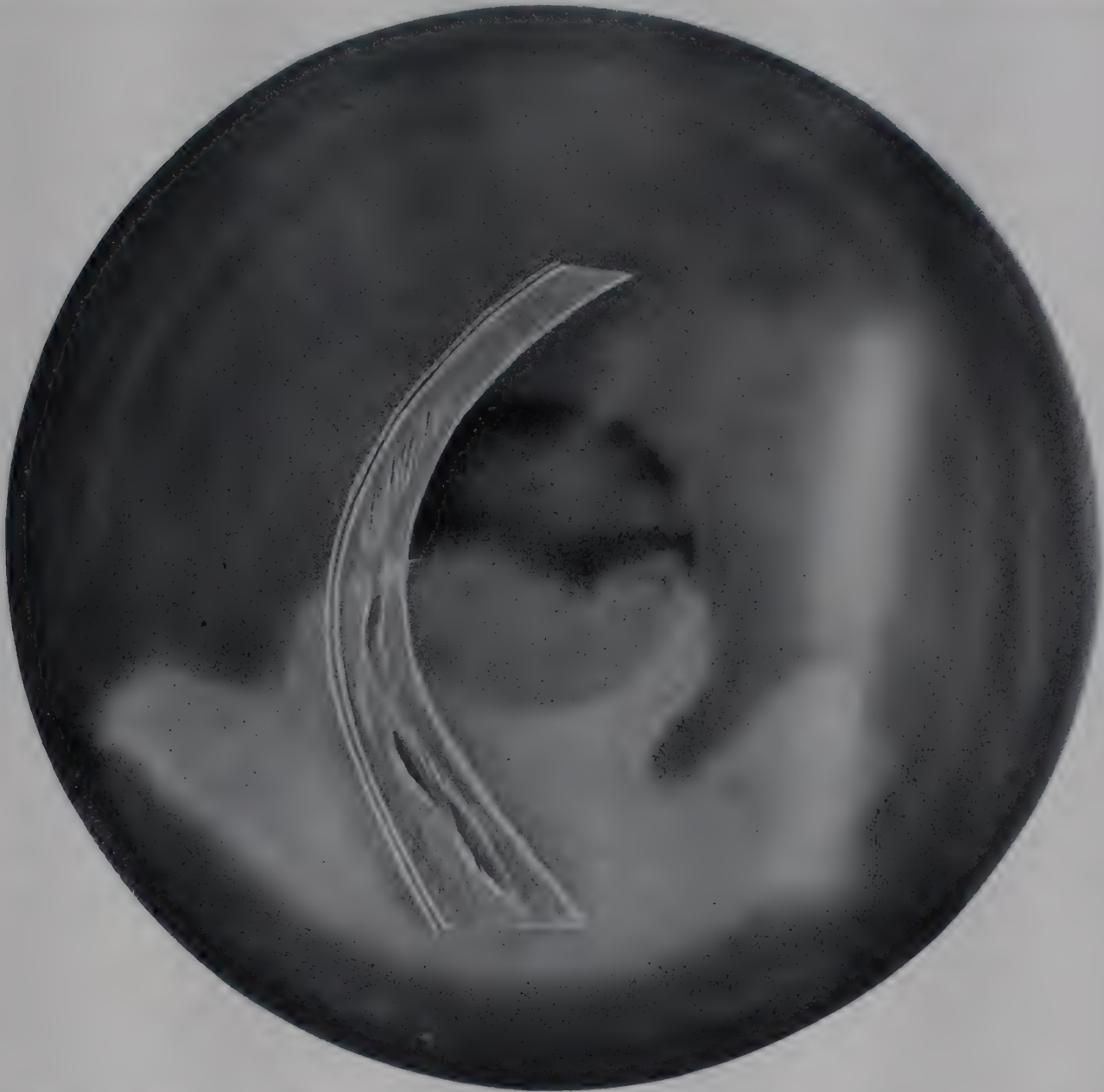


FIG. 190. Keratoconus. Optic section illustrating the thinning of the cornea at the apex and the change in the substantia propria consisting of short white striae. Note the dark, somewhat elliptical spaces, which are probably so-called "water slits" resulting from the ingress of aqueous. The lower part of the section is thicker owing to the presence of edema. The opaque area seen in lower part of the cornea by indirect illumination represents the extent of parenchymal edema.

adjoining parallel but stationary reflexes, when seen in specular reflection (page 422). By retro-illumination, these edges are dark. Deep fusiform or crescentic opacities, occurring at the summit of the cone, may follow ruptures in Descemet's membrane. Large ruptures may be associated with an "acute ectasia." Imbibition of aqueous through the damaged endothelium in the immediately neighboring corneal tissue follows; this is manifested by corneal

cloudiness (Fig. 190). A deep posterior bedewing is seen and if it is not too intense, defects of the endothelial layer can be observed in the zones of specular reflection. In most cases endothelial regeneration occurs. The edema subsides and the cloudy zone clears, the parenchyma regaining its original transparency. In others, permanent damage to the endothelium may result in scar formation. This causes localized fusiform or crescentic increase in relucency of the posterior corneal face.

(6) *Increased visibility of the nerve fibers.* Numerous investigators have noted that the nerve fibers in this condition become increasingly visible (Plate XXI, fig. 2). By direct focal illumination, a network of grayish lines, with corpuscle-like nodes at the point of branching are seen. Placing the focused beam (D. F. I.) at the corneoscleral junction, the proximal illumination so obtained at the limbus shows numerous spearlike nerve filaments entering the cornea. Since an increase in visibility of the corneal nerve fibers is observed with many other types of keratitis, and also, at times, in normal corneae, this finding cannot be considered as peculiar to keratoconus.

(7) *Fleischer's ring.* Fleischer⁸⁷ reported a pigmented line, partially or completely encircling the base of the cone. This line or ring, which varies from yellow-brown to an olive green in color, is found in Bowman's zone and has been attributed to the deposition of hemosiderin in fissures (Meesmann²²¹). It usually has the shape of a broken or interrupted ring, and only infrequently surrounds the entire cornea. It is covered by uninterrupted epithelium. According to Vogt, Fleischer's ring is identical with the "linea corneae senilis" (Stähli-Hudson line) (page 381). It is not found in every case of keratoconus, but when present it is best seen with high intensity of illumination. It is interesting to note that this ring, which is so arresting when seen with the biomicroscope, apparently has never been found in histologic preparations of keratoconus (Plate XXI, figs. 1, 2).

The biomicroscope is of great value in observing patients of keratoconus who have been fitted with contact lenses (Plates XXI,

fig. 3; XXII, fig. 1). The instillation of fluorescein assists in determining the points of pressure, especially at the sclerolimbic junction.

KERATOCONUS POSTICUS (BUTLER)

This condition was described by Harrison Butler⁴⁴ in 1930 as a rare anomaly of the posterior face of the cornea. It is characterized by increased curvature of the posterior corneal surface. The anterior surface remains unaltered. In consequence the cornea is thinner in the central portions than at the periphery.

Two varieties are known. In the first, a nonprogressive generalized regular increase of curvature occurs. Because of its resemblance to the shape of the fetal cornea, it is believed this type may be due to congenital arrest in development. The second type is more localized, almost disciform, usually in the center of the cornea, and appears as a regular cavity; it may result from trauma or from local corneal disease.²⁸²

MARGINAL DEGENERATION OF THE CORNEA (ECTATIC MARGINAL DYSTROPHY, PERIPHERAL FURROW KERATITIS, SENILE MARGINAL ATROPHY [FUCHS^{105, 109}], PERIPHERAL CORNEA ECTASIA [LAUBER])

This is a rare condition. It begins with a punctate infiltration in the anterior layers of the peripheral cornea, usually above, resembling arcus senilis and at times coincident with it; that is, there is a clear band between the infiltration and the limbus. The lesion may be bilateral.

The degeneration is characterized by early development of a superficial vascularization. Because of thinning of the subepithelial tissue (degeneration of Bowman's membrane and parenchyma) in the involved area, a depression of the corneal surface results, and leads to formation of a gutter-like furrow or trough. However, the epithelium remains unaffected. The furrow is bounded centrally by a steep wall, the sharply defined edge of which appears as a white irregular line. As the disease progresses, increasing degeneration of the subepithelial tissue is followed by thinning of the floor and deepening.

ing of the furrow. Vessels in the form of superficial radial branches invade the gutter, extending from the conjunctival and limbal arcades (Figs. 192, 193).



FIG. 191. Ectatic marginal dystrophy of the cornea.

Owing to weakening of the tissues, the involved portions give way before the intra-ocular tension and ectasia results (Plate XXI, fig. 4). Localized solitary or multiple ectatic areas may form, or the entire portion of the floor may become ectatic. Ectasia is usually limited to the upper portions of the cornea; but in rare instances, it may involve the entire circumference in varying degrees. This ectatic process is slow and may not be completed for one or two decades. Zentmayer³⁴² reported a case in which rupture of the floor of the groove occurred with consequent prolapse of the iris and incarceration. In Doggart's⁶⁰ case, studied biomicroscopically, optic section revealed marked thinning of the cornea throughout the

ectatic area. The endothelial mosaic was not visible in the center of the ectatic zone. Below the ends of the white line, there were numerous glistening crystals and opacities in the corneal stroma. The

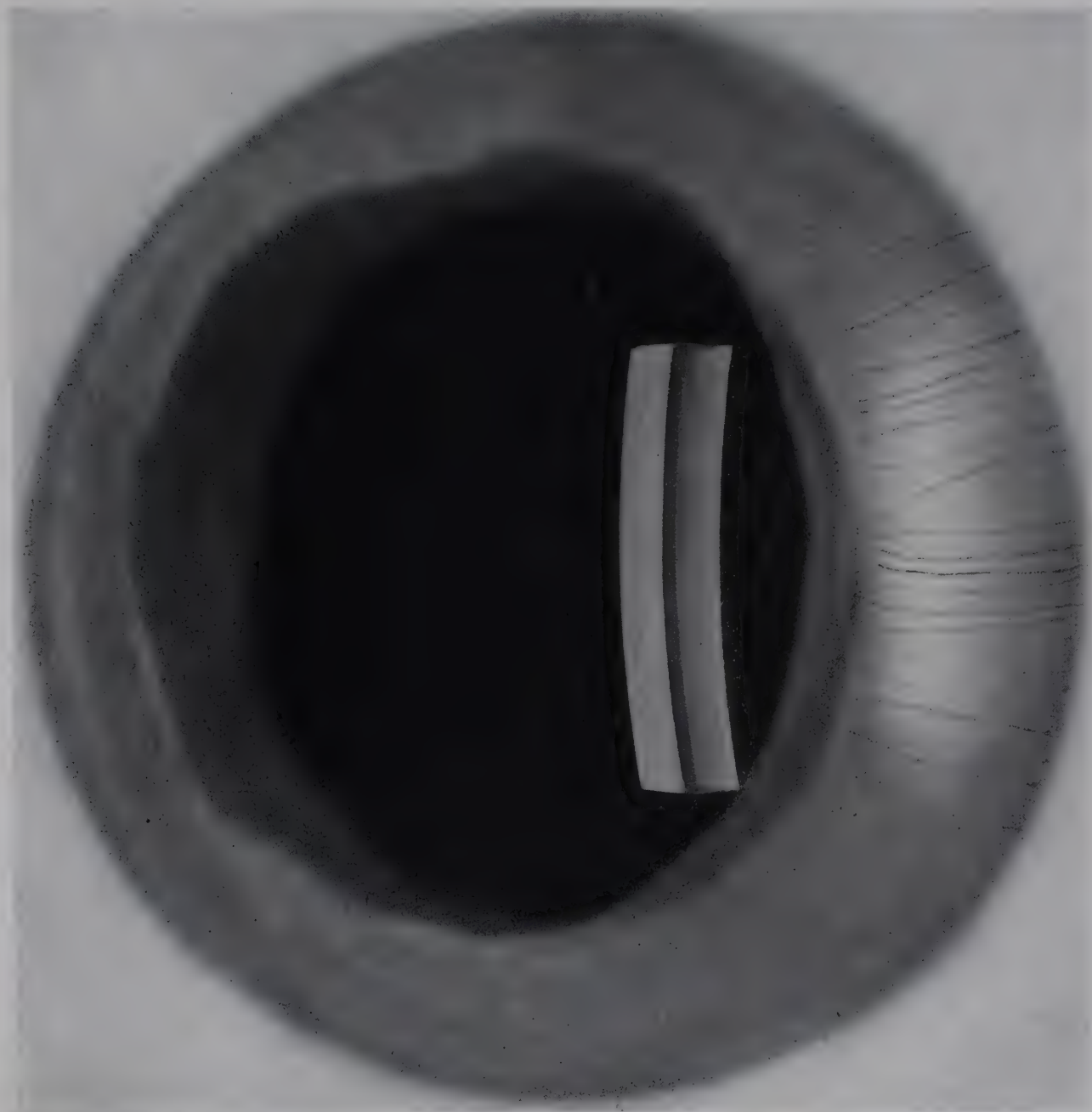


FIG. 192. Ectatic marginal dystrophy in the left eye. In the ectatic area to the right, vessels are seen by retro-illumination.

ectatic area was profusely vascularized. In the lower portion of this eye, there were many small grayish infiltrates in the parenchyma with beginning vascular encroachment of the limbal vessels.

In the aged, mild forms of a similar condition may occur, which never advance markedly and probably differ from the classical type. When these cases are examined with oblique illumination or with the parallelepiped, a furrow-like depression is seen at the limbus. When associated with arcus senilis, it occupies the so-

called "lucid interval." However, at times, in optic section, it may be seen that the loss of substance is only apparent because the groove



FIG. 193. Ectatic marginal dystrophy in the right eye of case shown in Figure 192. Optic section of the lower part of the cornea indicates the extreme thinning. The thinned out area of cornea is vascularized.

is filled in with transparent corneal epithelium (Plate XXII, fig. 2). In this case, the deeper, more relucet, furrow-like area is located in the subepithelial tissue.

WHITE LIMBUS GIRDLE

Vogt^{326, 327} described two types of whitish peripheral opacities at the limbus, one of which grossly may simulate arcus senilis. These occur chiefly in the interpalpebral regions. In the first type, as in arcus senilis, there is a clear interval between the opacity and the limbus (Fig. 194). With the biomicroscope, a structure vaguely similar to that of bandform keratitis (page 343) is seen. In the second type (Fig. 195), the opacity is continuous with the sclera, ensheathing

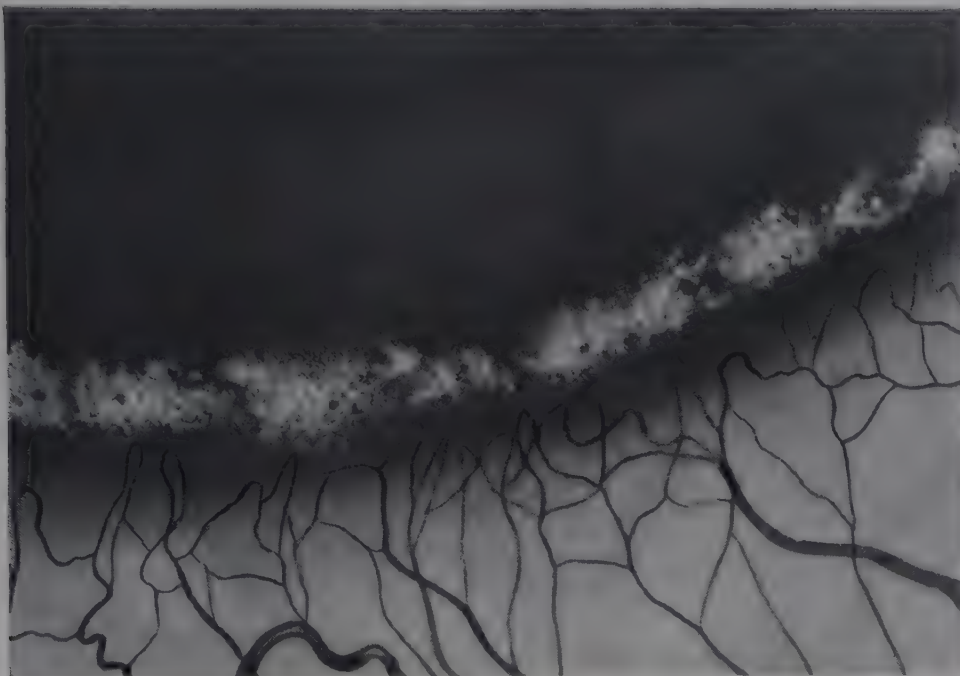


FIG. 194. White limbus girdle. Type I.

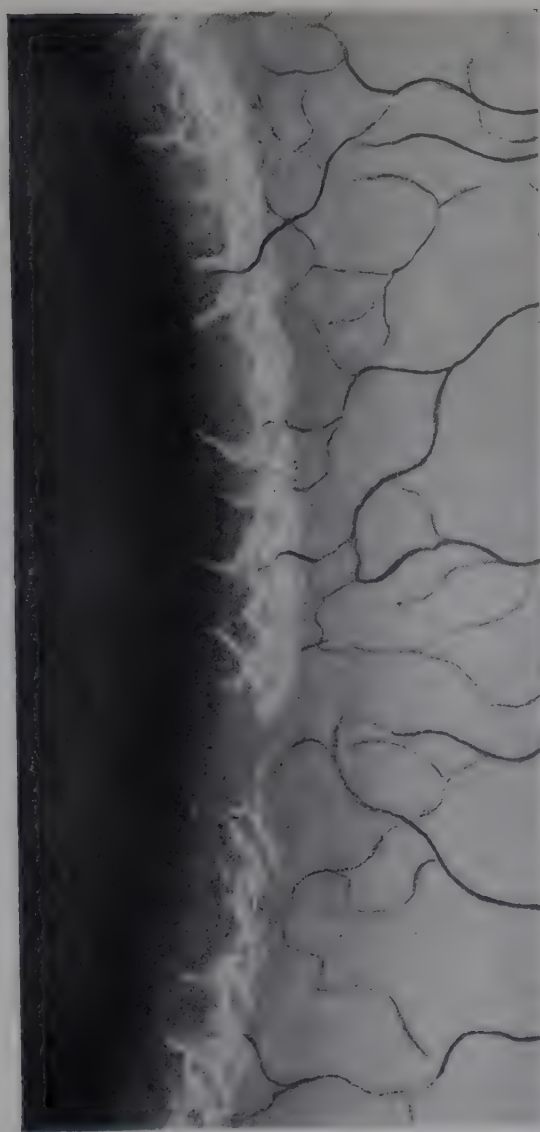


FIG. 195. White limbus girdle. Type II. (After Theodore.)

the capillary loops with radiating curved white lines that have a chalky appearance. These structures are superficial (subepithelial) and are usually bilateral. Their pathogenesis is unknown, but they may represent a dystrophic change, possibly on a hereditary basis.

HYALINE AND CALCAREOUS DEGENERATION

Hyaline and calcareous degenerations may occur in the cornea either as a primary change or secondary to chronic disease. The physical properties of hyaline are homogeneity and translucency. Because of its acellular structure and for other reasons it must be classified as a product of living cells, corresponding to a secretion. Pathologically, the deposition of this inert substance from living cells represents a form of necrobiosis. A hyaline-like secretory process seems to occur embryogenetically in the development of the extracellular glasslike membrane of Descemet, the lens capsule, and the lamina vitrea of the choroid. It may be that hyaline excrescences of Descemet's membrane and a rare type occurring in Bowman's membrane, described by Lugli,²⁰¹ represent a transition state between normality and pathology. Secondary hyaline degeneration frequently occurs in corneal scars and in pannus. A typical example of hyaline degeneration is seen in bandform keratitis and in the familial corneal dystrophies (nodular and reticular).

The deposition of calcium salts (carbonates and phosphates), resulting in the so-called calcareous degeneration, is usually secondary to fatty or hyaline changes. The lime is deposited in the form of granules or clumps. In the cornea, this usually occurs in the external layers (Bowman's zone and the superficial layers of the substantia propria). Superficial incrustations may become irritating and act as foreign bodies. When they become dislodged, as a result of sequestration, ulceration may ensue.

PIGMENTATION OF THE CORNEA

The following classification of pigmentation of the cornea is modified from that of Duke-Elder.⁴⁴

- I. Melanin
 - A. Epithelial and juxta-epithelial
 - 1. Congenital melanosis (racial)
 - 2. Age
 - 3. Associated with benign or malignant conjunctivo-limbal melanomas (Plate 13, fig. 1)
 - 4. Post-trachomatous pannus and xerosis
 - 5. Vortex dystrophy
 - 6. Ochronosis
 - B. Endothelial
 - 1. Congenital — associated with persistent adhesions of the pupillary membrane to the cornea
 - 2. Physiological: Turk's line, Krukenberg's spindle
 - 3. Degenerative: corneal dystrophies; age
 - 4. Traumatic or postoperative deposits
 - 5. Postinflammatory: keratic precipitates; uveal pigment
- II. Hematogenous pigmentation
 - A. Blood staining of the cornea (hematin)
 - B. Kayser-Fleischer's ring (?); Wilson's disease
 - C. Stähli-Hudson line (?) (epithelial)
- III. Metallic pigmentation
 - A. Argrosis: generalized or local
 - B. Chalcosis: copper
 - C. Siderosis: iron

Pigmentation of the cornea may occur in apparently normal eyes. In the human body, there are three known types of pigment, melanin, lipochromes, and the derivatives of hemoglobin. Each of these can be found in the cornea in various instances. In the region of the limbus, Redslob²⁴⁷ demonstrated the presence of potential melanoblasts. These cells contain a colorless substance, convertible into melanin pigment through the action of oxidizing ferment (dioxypyhenylalanine, "dopa"). This fact may explain many obscure corneal melanin pigmentations.

In addition to the aforementioned pigments, heavy metallic substances, for instance, silver (argyrosis), copper (chalcosis), and iron (siderosis), may cause staining reactions in the cornea.

MELANIN PIGMENT

This form of pigmentation is usually found on the posterior corneal surface (endothelium) congenitally, or following an inflammatory or degenerative process. Pure epithelial pigmentation is rare. Vogt²⁴⁵ described a case of superficial corneal pigmentation occurring in a man 53 years of age; the pigment was situated in the lower part of the cornea and resembled an incomplete arcus.

In dark races (Plate IV, fig. 3), the normal limbal pigmentation seen around the capillary loops may occasionally extend beyond the limbal margin into or below the corneal epithelium. In the aged (Plate II, fig. 5), in connection with the pigmentation surrounding the limbal capillaries, delicate granules of a melanin-like pigment may be present in the adjoining corneal epithelium. In severe degenerative corneal conditions, caused by trachoma or xerosis, epithelial pigmentation may also be observed. Although the pigment in these cases resembles melanin in character, a possible hematogenous origin must be considered, particularly when the cornea is vascularized. Epithelial and subepithelial pigmentation is also associated with limbal melanomas and conjunctival ochronosis. Pillat²⁴⁰ reported four cases of melanoma of the corneal epithelium. In these the pigment cells were disposed in groups in a linear or sector-shaped fashion, situated about 1 mm. from the limbus. The pigment extended toward the center of the cornea. There were no corneal vessels. The epithelial surface was not raised or uneven. No other ocular changes were noted. Because of the fact that such pigment groups could be found in an avascular tissue, like the cornea, Pillat concluded that melanoma and melanoblastoma are not of vascular or lymph origin.

Although pigmentation of the posterior corneal surface rarely occurs as a normal physiologic process in the young, it is not an uncommon finding in those of advanced age. It has been noted in old people suffering from severe systemic disorders (e.g., diabetes) and in association with cataract and glaucoma. It ranges from light yellow to dark brown in color and should be considered as a keratic

precipitate. The distribution likewise varies markedly in different cases, probably depending on convection currents in the anterior chamber, on the effect of gravity, and on the condition of the endothelium. Like inflammatory keratic precipitates, it is most frequently observed in the lower portion of the cornea, in a triangular or semilunar disposition. However, it may be seen as a thin vertical line (Turk's line)³¹² or the pigment may be deposited in spindle-shaped form (Krukenberg's spindle). Vogt has described a mosaic-like arrangement of the pigment granules on the posterior corneal surface, corresponding to the hexagonal outlines of the endothelial cells. I have seen this in a case of endothelial dystrophy. In pathologic conditions of the uveal tract — inflammatory, traumatic or degenerative — pigmentation of the posterior corneal surface is nearly always present, as independent granules, stellate melanophores, or a fine dust on keratic precipitates (Plate XXIX, fig. 6; Plate XXX, fig. 4). The finest pigment particles may appear faintly yellow or even gray in direct focal illumination. The ease with which pigment becomes dispersed in the anterior chamber is demonstrated by its presence on the posterior corneal surface after only mild inflammations, glaucoma, intra-ocular operations, or trauma. According to Barkan, blocking of the filtration angle, which may be observed by means of gonioscopy, is caused by dispersion of uveal pigment and may be an important factor in the pathogenesis of chronic simple glaucoma. Koeppe believed that a preglaucomatous state was indicated by the migration of groups of fine pigment granules (pigment epithelium) to the surface of the iris; this is especially visible in blue irides. The condition could also be a senile change but the question is as yet undecided.

Superficial Vortical Pigmentation (Whorl Opacity or "Dystrophy"). A rare type of superficial pigmentation, taking the form of converging, comet-shaped, whorl-like bands radiating to the periphery from a point near the center of the cornea, was first described by Fleischer.⁸⁹ Recently, Bloch³⁴ described a case which occurred in a 16-year-old girl. There was no history or evidence of ocular inflammation, nor was there a history of hereditary symp-

toms (Plate XXII, figs. 3, 4). The condition was bilateral. Afocal illumination revealed irregular stripes, starting in the periphery and converging like a vortex, the center of which was slightly below the middle of the cornea. Each radiating spoke was composed of minute brownish granules. Biomicroscopic examination showed that these granules were located just below the epithelium. Between the stripes there were channels of clear corneal tissue. The deeper layers of the cornea were normal and corneal sensitivity was not diminished. Vision was unimpaired. Vogels,³²⁰ who reported a case in 1931, believed that this was a rare type of corneal dystrophy. Following histochemical examination, he found that the deposit was due to glycogen. Bloch is of the opinion that these granules are composed of melanin; this is more in accord with the facts, as glycogen is not a normal constituent of the cornea.

Krukenberg's Spindle. Krukenberg¹⁸⁵ reported a bilateral endothelial pigmentation in the form of a vertical spindle. Its etiology is still unknown but originally it was considered to be a congenital anomaly. The spindle is composed of a dense grouping of fine pigment granules situated in front of the pupillary area (Plate XXII, figs. 5, 6). As a rule the spindle is small, its length usually ranging from 2 to 4 mm.; but it may be so large as to extend over the entire vertical diameter of the cornea. In many cases, with the biomicroscope, a more delicate pigmentation may be seen surrounding the main spindle. Vogt³²¹ described a case in which the deposition of the pigment granules in the spindle was hexagonal, forming a mosaic, closely resembling the endothelial cells in shape and size. Krukenberg's spindle, as a rule, is not associated with congenital (persistent pupillary membranes) inflammatory or degenerative defects of the iris. Most investigators agree that it appears after the second decade, especially in myopic females. Because of these facts, certain observers (Vogt,³²⁶ Koby¹⁷⁴) believe that it is not congenital but acquired. Vogt maintains that it is only a phase of senile or presenile pigmentation. Koby believes that it is due to the mobilization of pigment from the ciliary body rather than from the iris, and that like other precipitates its presence and arrangement depend on

currents in the anterior chamber and endothelial alterations. He cites a case in which a spindle formed in one eye following iridocyclitis.

HEMATOGENOUS PIGMENTATION

Blood Staining of the Cornea. Blood staining of the cornea may occur (a) from extracorneal sources and (b) from intracorneal sources. In the first case, it usually results from long-standing hyphema in the anterior chamber. The slow absorption of massive or recurrent hemorrhages in the anterior chamber following surgical procedures in the presence of pathologic iris vessels, or following trauma, especially when there is increased intra-ocular tension, may result in migration of blood pigment into the deeper corneal layers. Traumatic ruptures of the iris are usually accompanied by bleeding into the anterior chamber. Spontaneous hemorrhage in the anterior chamber may occur with intra-ocular tumors or following thrombosis of the central retinal vein.

After blood has filled the anterior chamber for some time, the cornea suddenly assumes a hazy color, varying from a dirty gray to a greenish black or brown. The entire cornea may be involved but more commonly there is a discoid opacity in front of the pupillary zone.

Plate XXIII, fig. 4, illustrates a case of so-called spontaneous anterior chamber hemorrhage. There was corneal staining on the posterior face following iridic hemorrhage, secondary to occlusion of the central retinal vein. The intra-ocular tension was increased. The lower part of the anterior chamber was occupied by a bright red hyphema; from either side an irregular band of blood encircled the periphery of the anterior chamber extending into the filtration angle. Engorged vessels traversed the iris in a circular manner, branching superiorly like a brush. Bright red blood cells were seen below on the endothelial surface, while above reddish brown blood pigment was precipitated. There was increased reluctance of the beam as it passed through the anterior chamber, which under high magnification showed fine brownish floating dots. Immediately fol-

PLATE XXII

FIG. 1. Optic section through cornea with contact glass in position. Foreign particles in buffer solution containing fluorescein between cornea (to the right) and contact glass (to the left) are stained yellow. The cornea has a yellowish brown appearance. The back part of the contact glass is stained green.

FIG. 2. Senile marginal degeneration of the cornea. Optic section reveals that defect in the stroma has been filled in by epithelium. The area occupied by the epithelium appears dark behind the film line.

FIG. 3. Vortex dystrophy. Showing whorls of pigment in characteristic arrangement in a young girl, 16 years old. Diffuse illumination. (After Bloch.)

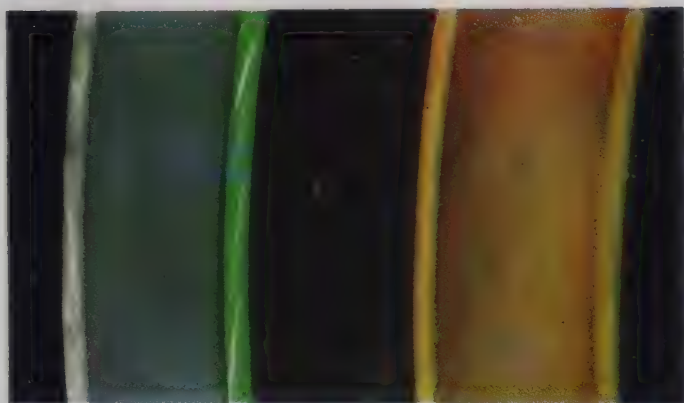
FIG. 4. Vortex dystrophy. Direct focal illumination. Showing character and superficial deposition of the pigment.

FIG. 5. Krukenberg's spindle. Vertical fusiform distribution of pigment as seen with the ophthalmoscope.

FIG. 6. Krukenberg's spindle. Appearance and localization of pigment on the posterior corneal face. Direct focal illumination.



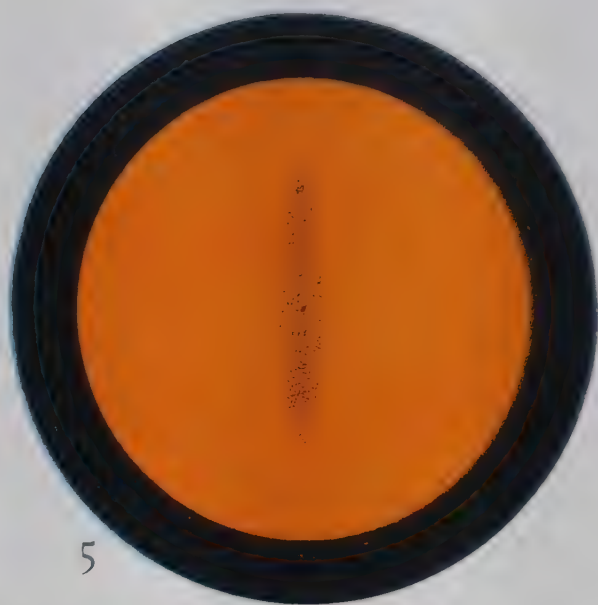
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lowing paracentesis to reduce tension, the entire anterior chamber filled with blood; several weeks later this blood was partly absorbed, leaving a typical picture of corneal blood staining. The resulting appearance was similar to that shown in Plate XXIII, fig. 6, which occurred in another case from engorged iridic vessels where the central area of the cornea was occupied by a grayish brown disklike infiltration, resembling, at a casual glance, a dislocated lens. In optic section, the disk consisted of a grayish brown haze in the deeper layers, which under high power was seen to be punctate in character. The cornea was thickened in this area. The epithelium over the disk was edematous. Delicate brownish dots of pigment were strewn over the posterior face.

Such an opacity tends to clear slowly from the periphery inward; complete clearance, if it occurs, may take from one to two years. In other instances, following hyphema in the anterior chamber, red blood cells may arrange themselves in the form of vertical columns on the posterior corneal surface (similar to Turk's line), extending upward from the hyphema for several millimeters (page 577) (Plate XXIII, fig. 5). In Plate XXIII, fig. 1, is shown the migration of blood pigment cells across the cornea in a linear fashion following a subconjunctival hemorrhage after contusion. Optic section (Plate XXIII, fig. 2) revealed that the blood pigment had evidently migrated across the cornea in a rent in Bowman's membrane.

Peripheral staining of the cornea may occur following severe subconjunctival hemorrhage. A golden brownish pigmentation may be seen even in the deeper layers of the cornea, separated from the conjunctival hemorrhage by a clear space (Plate XXIII, fig. 3).

Intracorneal blood staining occurs from rupture or disease of vessels which are present as part of a pathologic process, as in interstitial keratitis. In these cases, small bright red areas may be present for a long time before disintegration of the red cells occurs and before secondary deposition of pigment, derived from hemoglobin, results. Staining from blood pigment must be distinguished from the siderosis associated with intracorneal iron foreign bodies.

Histologically, in blood staining of the cornea, the products of

PLATE XXIII

FIG. 1. Linear migration of blood pigment in Bowman's zone following subconjunctival hemorrhage due to trauma.

FIG. 2. Optic section through pigment line seen in Figure 1 showing location of pigment on the surface of Bowman's membrane.

FIG. 3. Blood staining in corneal parenchyma following subconjunctival hemorrhage. Direct focal illumination.

FIG. 4. Blood staining of cornea following hyphemia in a case of thrombosis of a central retinal vein and rubeosis glaucomatosa. Blood pigment appears in the anterior chamber flare.

FIG. 5. Vertical arrangement (columns) of red blood cells on posterior corneal surface with hyphemia.

FIG. 6. Grayish discoid corneal opacity following anterior chamber hemorrhage, simulating an anterior dislocation of the lens.



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the degenerated blood corpuscles, pigment granules and precipitated albumin may be present²³⁵ in and between the corneal corpuscles and lamellae.

Stähli-Hudson Line. The presence of this superficial brownish line, which was first noted by Hudson,¹⁵⁴ was more extensively described by Stähli,²⁸¹ independently, in 1918; it is the latter's name that now commonly designates this condition. The line generally has a horizontal direction and is usually situated in the interpalpebral space in the lower half of the cornea. As a rule, it never extends into the limbus; it is wavy, tenuous, and may be branched (Plate XXIV, figs. 1, 2).

The line, as originally described, occurred in the normal cornea of the aged (*linea corneae senilis*); later it was noted in pathologic corneas (scars) of patients of various ages. Vogt demonstrated that the earlier description of its color was erroneous, because insufficient illumination was employed by the examiners. With the arc lamp, he differentiated three types of staining: (1) Bottle green to olive yellow type, having a homogeneous lacquer-like appearance, with poorly defined borders. This is the most common type. This line has a width ranging from 0.05 to 0.15 mm. and a length of several millimeters. (2) Ochre yellow type, in which the line is thin but sharply outlined and granular in character. (3) Colorless type, first described by Vogt as having a white or grayish appearance with grayish extensions which may be mistaken for scars.

The latter two types can be seen only by retro-illumination. In addition to the forms described, a single line may show all three color variations. It should be emphasized that since the lines may appear in the apparently normal eyes of the aged, they should be known as "superficial senile lines."

Besides the aforementioned senile type, pigmented lines have been noted over corneal scars in patients of all ages. These lines resemble Fleischer's ring, which occurs in keratoconus. I have observed them frequently in many cases of scars or leukomas. Irregular pigmentation occurs on the surface of such lesions. They are best seen in

PLATE XXIV

FIG. 1. Stähli-Hudson line. 60 \times .

FIG. 2. Stähli-Hudson line (60 \times) with areas of calcareous degeneration and superficial vascularization. (Diffuse illumination.)

FIG. 3. Stähli-Hudson line. Details in optic section. A superficial corneal scar crossed by a greenish brown pigmented line; deep vessels and branching corneal nerve above.

FIG. 4. Kayser-Fleischer ring. (Diffuse illumination.)

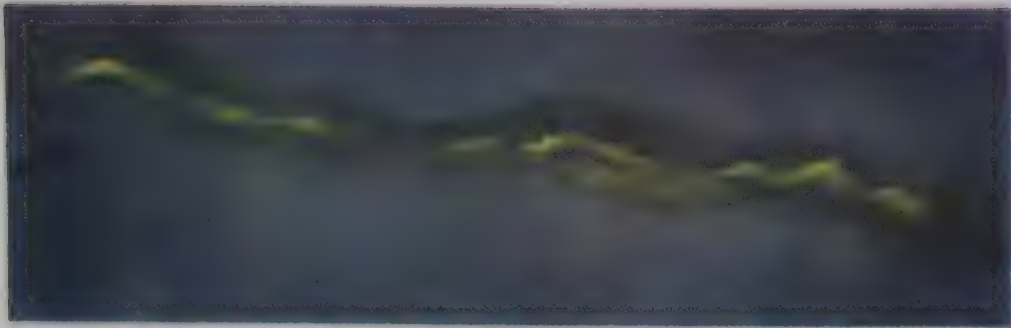
FIG. 5. Kayser-Fleischer ring in direct focal illumination; the deposition of pigment is in Descemet's zone. Color seen by retro-illumination. (After Gartner.)

FIG. 6. Kayser-Fleischer ring. Direct focal and retro-illumination. So-called sunflower cataract on the anterior lens capsule. (After Gartner.)

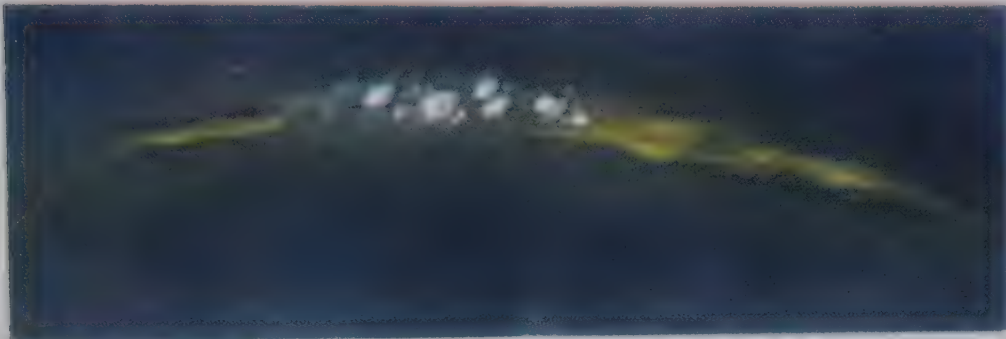
FIG. 7. Copper deposits in Bowman's zone following prolonged use of copper sulphate stick for the treatment of trachoma. (After Meesmann.)

FIG. 8. Copper deposits in Descemet's zone in case of a penetrating foreign body (copper particle).

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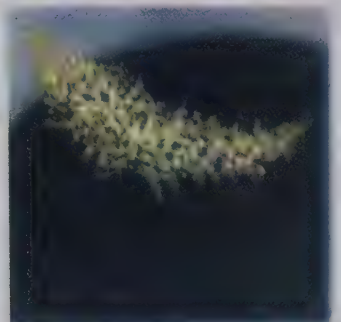
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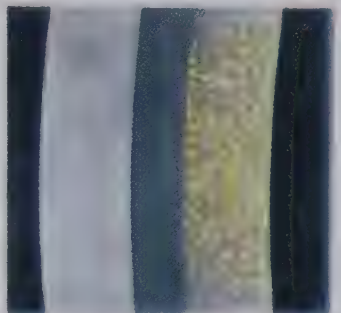
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strong direct focal illumination and do not always take the form of a line but may be irregularly shaped (Plate XXIV, fig. 3). They never completely cover the nebulous area and are usually situated in the center or near the edge of the scar, corresponding to the place where the lower eyelid touches the cornea. In optic section, the pigmentation is seen below the tear film line, close to Bowman's zone; only when the epithelium is raised or edematous does surface irregularity result (Plate XXIV, fig. 3). According to Vogt these lines at times may have a transient character. He mentioned the appearance of a pigmented line in a case of keratitis eczematosa; the line disappeared completely after two years.

The etiology of these structures is still not understood, although various explanations for their appearance have been suggested. The pigment has been shown to be hemosiderin.^{226, 281} Stähli,²⁸¹ who studied this line chemically as well as histologically, found that (1) it is situated intracellularly in the epithelium; (2) it is composed of small granules; and (3) it is an alkaline hematin. The presence of such a line, even in avascular scars, has aroused some question as to the nature and mechanism of the pigment impregnation. Stähli believes that the pigmentation results from an impregnation of iron, which is normally found in the tear fluid. It is possible that these lines may be caused by degenerations or tears in Bowman's membrane due to traction, followed by secondary pigmentation. This would be in accordance with their irregular granular appearance.

Kayser-Fleischer Ring. An important diagnostic sign in hepatolenticular degeneration (Wilson's disease) is the Kayser-Fleischer ring. This ring is of arresting appearance and occupies the periphery of the cornea, being restricted to the deepest layers. It may be visible to the unaided eye.*

* In a personal communication Dr. Samuel Gartner described the Kayser-Fleischer ring thus:

"With ordinary illumination it seems as if only the limbus were outlined by a dark, almost black, circle. With stronger, focal illumination, the ring in the cornea is green-brown. With the slit lamp it is a brilliant golden brown, resembling the tint of a maple leaf in autumn. The outer part is densely pigmented, with some mottling. The inner part is composed of more discrete granules of pigment.

"The inner border has a transition zone of from 2 to 3 mm. between it and the clear cornea. This zone is tinted a delicate blue, with slight mottling. I found this in all the cases I observed, though it is not generally described. This blue zone has added interest as its color and quality

The colors of the ring which vie with those of the spectrum, range from violet to red; but olive green and yellow usually predominate. The hue may change depending on the type of illumination employed. In Plate XXIV, fig. 4, it will be seen that by diffuse illumination the ring has an olive-green hue. In direct focal illumination, it appears grayish, while by retro-illumination it has a reddish coloration (Plate XXIV, fig. 5). It lies adjacent to the limbus and spreads over the posterior face, usually being from 1 to 3 mm. in breadth. A second ring may form within the peripheral one.

A case of Kayser-Fleischer ring reported by Poe,²⁴⁴ which I examined biomicroscopically, was typical. With the unaided eye, there was a bilateral brownish green ring, encircling the cornea at the limbus. With the biomicroscope, the pigmentation was observed on the posterior face of the cornea. Its hue varied from a peripheral brownish green through orange to yellow in its central portion. The pigment was granular in appearance. The entire ring was about 2.5 mm. in width; in places there was a clear interval between it and the limbus. Exact localization showed its distribution to be just in front of the endothelium in Descemet's zone.

In some cases, the ring may be more prominent above and below, thinning out to the sides. Frequently, the so-called "sunflower cataract" is found in association with the Kayser-Fleischer ring. This appears as a discoid discoloration (greenish yellow) with or without stellate extensions on the anterior lens capsule (Plate XXIV, fig. 6).

There is still considerable doubt as to the chemical nature of the infiltration. One school of thought^{201, 212, 214, 242} believes that it is due

resemble those of the lens opacity found in 1 of my cases and described by others as the sunflower opacity. Whether the blue zone is a second type of pigment or the same as the rest of the Kayser-Fleischer ring in a thinner layer I could not determine. I am rather inclined to believe that it is a thinner layer and therefore gives a different optical effect.

"Six cases have been described in which, in addition to the Kayser-Fleischer ring, there was an opacity on the anterior capsule of the lens. This was seen in both eyes in one of my cases. The opacity was about 4 mm. wide and occupied the pupillary portion of the lens, while the periphery was clear. Some patients had extensions of the opacity in a petal-like arrangement (sunflower cataract). This type of opacity is not a cataract, and the name sunflower cataract is a misnomer. It is a pigment deposit on the lens capsule. Under the slit lamp the opacity is an extremely thin layer of pigment on the anterior capsule, tinted a delicate blue; the surface is granular and the edges are fuzzy."

either to silver (endogenous argyrosis) or to copper deposits secondary to failure of elimination of these elements. However, Gerlach and Rohrschneider¹¹⁵ were unable to demonstrate the presence of silver by spectroscopic methods. Another school believes that it is hematogenous in origin. Poe¹⁸⁸ stated that "the material, producing the corneal changes in hepatolenticular degeneration, represents derivatives of the bile pigments, which are washed into the circulation in the course of hepatic cirrhosis. These same substances, that are active in the ocular tissues, in all probability, are responsible for the degenerative processes that occur in the lenticular nucleus of the cerebrum."

METALLIC PIGMENTATION *

Argyrosis of the cornea (Plate XXV, fig. 1) occurs in its deeper layers (Descemet's membrane). This fact has not been recognized widely, because in mild forms it is impossible to see the deposit without the aid of the biomicroscope. Argyrosis usually follows prolonged local application of silver nitrate or instillation of colloidal silver compounds. Some rare cases of ocular argyrosis have been reported following intensive administration of silver preparations internally. Friedman and Rotth⁹⁷ observed corneal involvement in ten of twelve patients with conjunctival argyrosis. As previously mentioned (page 174), silver is chemically united with the tissue by forming an albuminate compound. In the cornea reduction occurs in the elastic fibers of Descemet's membrane. This reaction may be influenced by light. The silver passes through the layers of the cornea, leaving no visible trace, until it reaches Descemet's membrane, where

* Regarding chrysiasis (gold impregnation) of the cornea, Givner says (personal communication): "Slit lamp examination of approximately twenty-five patients receiving intra-muscular gold injections failed to disclose any deposits. Recently, upon the use, as a routine in the arthritis clinic of Solganal B. Oleosum (a suspension of Aurothioglucose in Sesame oil containing 50% gold), three patients were seen with fine punctate glistening deposits under the epithelium. One patient had both corneas studded with these crystal-like deposits. In the other two cases, one eye was more involved than the other. There were no deposits seen near Descemet's membrane as has been reported by Lisch.

It is quite possible that the beginning of these gold deposits are an index of the therapeutic saturation point of other tissues.

An unsuccessful attempt to produce this condition in a rabbit was made. The attempt was undertaken with the idea of removing the corneas and determining the nature of the deposit chemically."

PLATE XXV

FIG. 1. Argyrosis of the cornea; staining of Descemet's zone in a case having marked argyrosis of the conjunctiva.

FIG. 2. Argyrosis of the cornea showing involvement in Descemet's zone (direct focal illumination) by higher power.

FIG. 3. Epithelial edema (bedewing) ($40\times$) by direct retro-illumination showing carpet of vacuoles (focused at anterior corneal surface).

FIG. 4. Same case (Fig. 3) with posterior corneal surface in focus showing keratic precipitates. The epithelial droplets are now out of focus.

FIG. 5. Edema of corneal epithelium in glaucoma. Direct focal and retro-illumination.

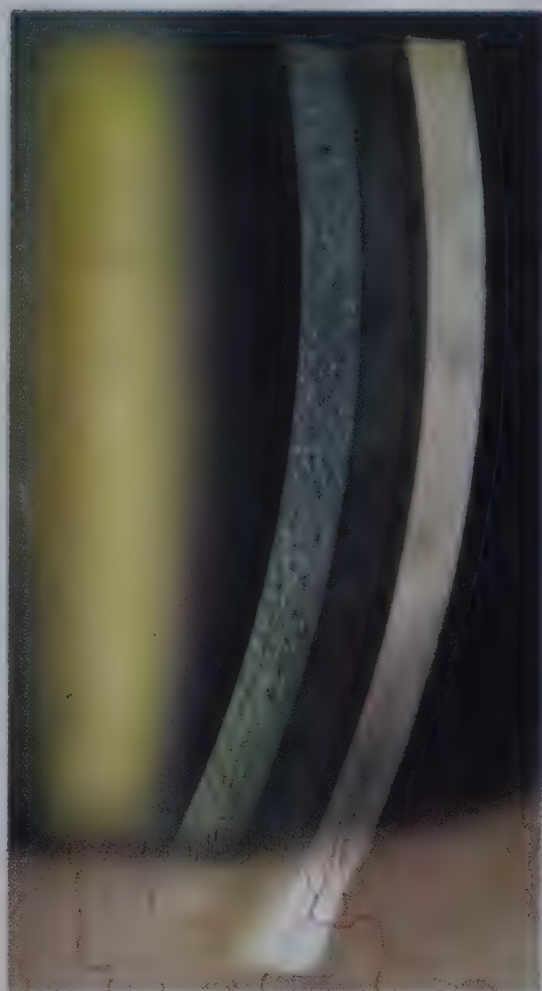
FIG. 6. Edema of corneal epithelium. Same case as shown in Figure 5. Large droplets, varying in size seen near the limbus by retro-illumination. $40\times$.



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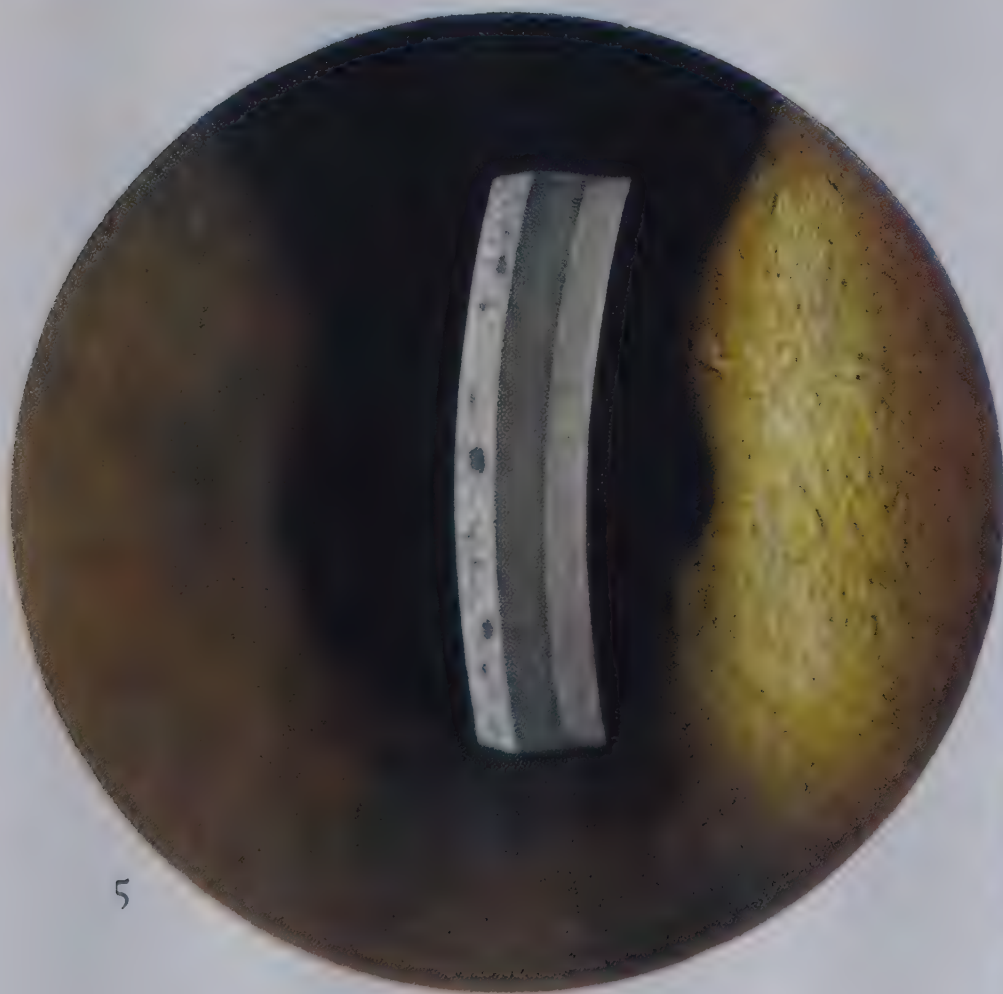
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precipitation occurs. This elastic hyaline structure has a peculiar affinity for heavy metals. The underlying endothelium is unaffected. The posterior face of the parallelepiped assumes a characteristic bluish or slate gray appearance, which is unique and pathognomonic. It can be likened to a light bluish moirée (Plate XXV, fig. 2) or stippling. In optic section, the deposition of the metal is seen to occur just anterior to the endothelial layer. The yellowish tinge, which this condition sometimes shows near the limbus^{97, 325} may be caused by interference phenomena. The extent of the pigmentary deposits varies from case to case. In some, the entire cornea may be involved, while in others it may be localized to certain sectors. Retro-illumination does not reveal its presence unless it is marked.

Siderosis. Iron particles embedded in the cornea, if not promptly removed, may leave a ring of granular pigment in the form of rust-like deposits, which results from the oxidation of the metal. This process is rapid and frequently on the day following the removal of the iron particle, a ring of "rust" surrounds the site of the embedded foreign body. By direct focal illumination, a ring of fine brownish granules is observed about a grayish white necrotic center. The cornea is usually unaffected in siderosis bulbi, but Wolff³³⁸ has reported a case in which corneal involvement occurred.

Chalcosis (χαλκος — copper). Copper impregnation of the cornea occurs following embedding or intra-ocular penetration of copper particles (Plate XXIV, fig. 8). In the latter case, it appears as a greenish or greenish yellow ring in the deeper layers of the cornea near Descemet's membrane, especially at the limbus. Locally, particles of copper embedded in the corneal parenchyma may be well tolerated or the surrounding areas may have a punctate deposition.²⁸⁵ It has also been reported following prolonged treatment in cases of trachoma with copper sulphate.²⁵⁹ Sallmann found an impregnation of copper in front of Descemet's membrane. Meesmann,²²⁰ in his atlas, shows a case of corneal chalcosis following the use of a copper stick. There were three sickle-shaped areas of a generalized yellow infiltration some distance from the limbus in Descemet's zone (Plate XXIV, fig. 7).

Chapter Thirteen

BIOMICROSCOPY OF CORNEAL CHANGES

THE avascularity and transparency of the cornea permits biomicroscopic study of the earliest pathologic alterations to a degree not possible in other tissues. In order to understand these changes it must be remembered that the normal cornea is a gel which in direct focal illumination exhibits a characteristic Tyndall phenomenon but at the same time by retro-illumination is non-obstructive. The slightest edema or minutest infiltration causes immediate changes in transparency and as already mentioned (page 74), when these changes are illuminated, they evoke certain optical phenomena.

The corneal surfaces are guarded by the epithelium and endothelium with their respective cuticular membranes. These layers, like skin, not only are protective but also play a role of utmost importance in controlling the normal physiologic functions of the underlying tissue. Alteration in colloidal properties and in water content leads to increased dispersion and reflection of light and augmentation of the Tyndall phenomenon (i.e., increase in relucency).

To appreciate all minute and early corneal alterations, repeated and even daily biomicroscopic examination are necessary. Needless to say, familiarity with the normal appearance of the cornea is a prerequisite to biomicroscopic diagnosis of corneal changes. All methods of illumination should be employed. Optic section (narrow beam) is invaluable. Fluorescein is the most satisfactory dye for demonstrating, in optic section, the stained precorneal film line which accentuates the underlying dark nonrelucant epithelium and the relucant line of Bowman's membrane. The normal parenchyma,

in direct focal illumination, presents the familiar relucet parallelepiped, in which minute reflexes (internal specularities) appear as the surface zones of specular reflection are approached. In addition, the configuration of the normal limbus must be borne in mind. In optic section, Bowman's membrane merges with the wedge of the "relucet superficial limbal spur" (Plate I, fig. 5), which represents the transition zone between the cornea and sclera. Arcades of the perilimbal capillaries extend over this area. Only when all these normal features are thoroughly understood is one able to recognize and pass judgment biomicroscopically on minute corneal changes of any kind. Finally, one must keep in mind that it is the optical phenomena secondarily effected by tissue changes that are actually being observed. Since these are incompletely understood false interpretations can easily be made; many observations and deductions that are now considered true may be changed and corrected in time.

It should be noted that the alterations enumerated in the following list are nonspecific. They may be found following trauma, in allergy and in the so-called degenerations, as well as in the definitely inflammatory lesions of the cornea. This, of course, is due to the fact that the anatomy and physiology of the avascular transparent cornea can only react according to certain well-defined patterns. The presence even of minute changes in the dystrophies and degenerations may evoke secondary inflammatory reactions (i.e., edema, infiltrations, and vascularization), which may mask the original picture.

EPITHELIAL CHANGES IN THE CORNEA

Corneal edema is one of the most frequent findings in inflammations, following trauma, in degenerations, and in the presence of increased intra-ocular tension. Any interference with the antidromic impulses of the sensory corneal nerves (e.g., neuromparalytic keratitis) may lead to epithelial edema and formation of vesicles. This has been explained as being due to the resulting accumulation of cellular metabolites (histamine-like substances).

The edema may be local or general, depending on the location and severity of the disease process. Edema of individual layers, that is, epithelium or endothelium, may occur without apparent involvement of the parenchyma. However, parenchymal edema may be associated with edematous changes of the epithelium. Edema of the parenchyma is best exemplified in the acute stages of interstitial keratitis (see page 449) and disciform keratitis, and with herpetic keratitis. Edema of the epithelium is manifested by the appearance of fine droplets or dewlike changes; while in the parenchyma it produces at first a diffuse haze and later, water-clefts.

EDEMA OF THE EPITHELIUM (BEDEWING)

The presence of edema in the epithelium or endothelium, whatever the cause, is known as "bedewing." This implies a misty appearance like a condensation of water vapor from the atmosphere on a glassy surface (Plate XXV, figs. 3, 4). The existence of such droplets in the epithelium does not necessarily presuppose inflammation, because this change can always be seen physiologically at the limbus in the region of the arcades, and it is a common finding in glaucoma. In this volume, the terms "bedewing" and "edema" are used interchangeably to describe a condition of droplet formation, which appears only in a single plane (i.e., in the epithelium or endothelium). Edema of the parenchyma does not give a dewy appearance because the morphology of the tissue is different. The droplets vary in size depending on the condition present. At the limbus, in the physiologic state, the droplets are finer, separated, and not easily recognizable (Fig. 156; Plate II, fig. 4). In glaucoma the droplets vary in size and are set closely together, forming a veritable carpet of easily recognized droplets (Plate XXV, figs. 5, 6). In inflammatory conditions like interstitial keratitis, the droplets are smaller and more uniform.

The readiness with which the epithelium becomes edematous may be explained by its anatomy and physiology. In addition to the function of protection, it controls the water balance and respiration (gaseous exchange) of the cornea. The surface of the epithelium

itself is protected by the fluid of the precorneal film. Any condition which prevents normal physiologic passage of fluids or gases through the epithelium causes stasis and resultant epithelial edema. Epithelial edema is associated with a host of superficial corneal conditions, such as certain corneal dystrophies, herpetic keratitis, keratitis eczematosa, ulcers, erosions and abrasions, the presence of foreign bodies, the instillation of cocaine (Fig. 163c).^{*} Severe conjunctivitis may evoke peripheral edema of the corneal epithelium. It occurs with intra-ocular conditions, such as glaucoma, inflammations of the uveal tract, and with the attending degeneration following these conditions. In these cases, edema is usually uniformly spread over the entire cornea, although localized epithelial edema may be observed in limited areas, for instance, over keratic precipitates. To the unaided eye, epithelial edema appears as a dull gray relucet haze, interfering with corneal luster. *Edema of the epithelium is best seen by retro-illumination.* By this method, the individual droplets are seen as a carpet of vacuoles of various sizes in one plane. Using the iris as a reflecting screen, the droplets appear brighter than the dark background (respersive phenomena is likewise seen in indirect retro-illumination) (Plate XXVI, fig. 2). The effects of epithelial edema may also be seen in the anterior zone of specular reflection (Plate XXVI, fig. 1). There is an increase in irregular reflections, caused by unevenness of the corneal surface. The appearance of these irregular reflexes in the zones of specular reflection is enhanced during the examination because of drying of the corneal surface. However, eyelid movements, causing a fresh flow of conjunctival moisture, may result in their transitory disappearance. By proximal illumination edema of the epithelium may also be seen close to the parallelepiped because of the scattered light. In direct focal illumination, when the edema is marked, it causes a peculiar irregular grayish pitted design on the surface of the parallelepiped.

Coalescence of these droplets or vacuoles leads to the formation of vesicles; on confluence these in turn may form large raised bullae

^{*} Cogan⁴⁹ believes that corneal edema results from relative hypo-tonicity of the fluid bathing the cornea and that the changes occurring in the epithelium with cocaineization are due to this rather than to direct toxic action of the drug itself.

PLATE XXVI

FIG. 1. Edema of corneal epithelium in glaucoma as seen in specular reflection.

FIG. 2. Epithelial edema. To the left, carpet of small vacuoles in case of cyclitis is seen by direct retro-illumination. Larger dark spots represent keratic precipitates or droplets. To the right, large whitish keratic precipitates are seen on the posterior surface of the parallelepiped.

FIG. 3. Corneal bullae in specular reflection.

FIG. 4. Corneal bullae by retro-illumination.

FIG. 5. Superficial vascularization associated with varicosities of the superficial conjunctival vessels in a case of nevus flammeus of the face and glaucoma (Sturge-Weber syndrome).

FIG. 6. Deep and superficial vascularization of the cornea near the limbus. Deep terminal loops and interanastomosing superficial capillaries in a case of interstitial keratitis. Upper part of the cornea (not shown) had numerous parenchymal infiltrates.



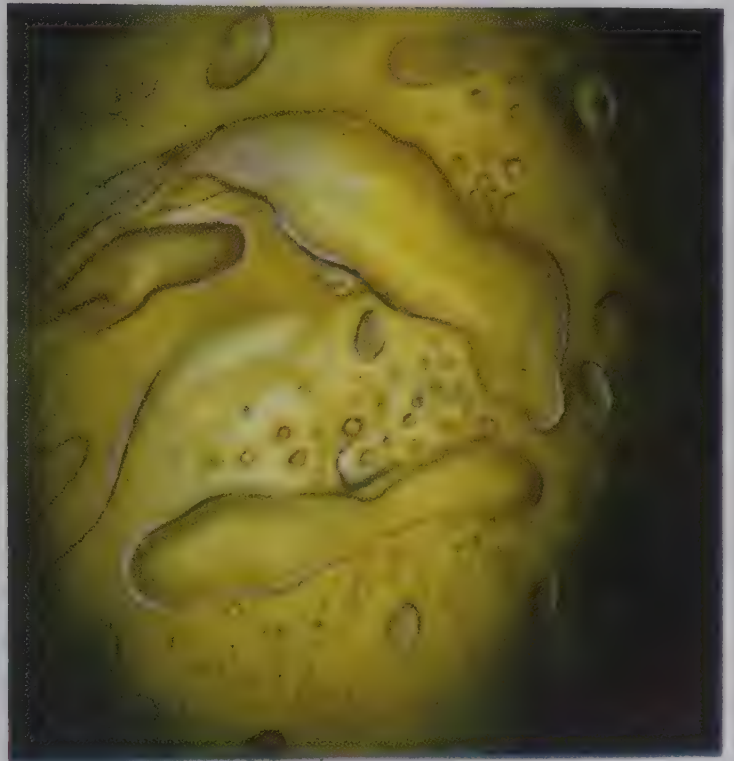
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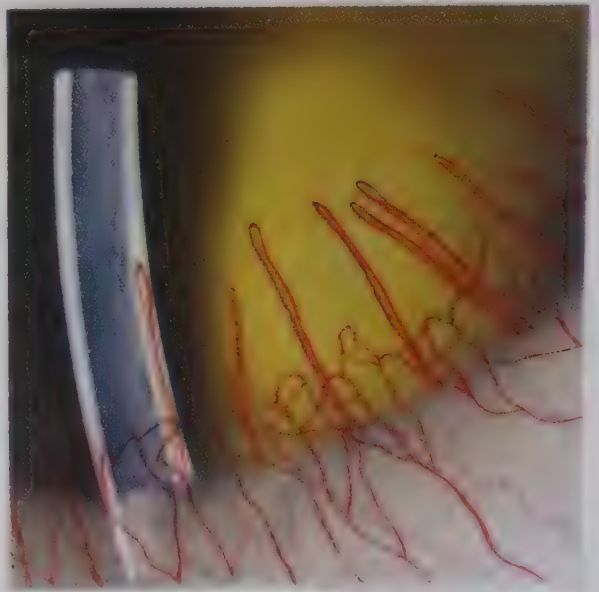
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(bullous keratitis). These vary in shape and size. Bullae are frequently seen on the corneae of degenerated globes. Although they may attain great size, there is little tendency for them to break



FIG. 196. Epithelial bullae (bullous keratitis). A. Appearance of bullae by direct focal illumination (high power) in a case of phthisis bulbi. B. Optic section through center of bullae, showing raised surface and clear contents.

down spontaneously (Plate XXVI, figs. 3, 4). When of sufficient size the vacuoles or bullae exhibit "unreversed illumination" by retro-illumination, that is, the border of the droplets nearer the reflected light is brighter than its opposite side. Furthermore, the phenomenon of primary distortion of the optic section, which

traverses the bullae, and secondary visual distortion of deeper corneal details (which is present when the axis of observation passes through the bullae) are noticed (Figs. 108, 109, 110). Instillation of fluorescein and observation with optic section aids in the delineation of raised vacuoles or bullae (Fig. 196). In the parallelepiped the staining of the precorneal film may also reveal stippling of the epithelial surface due to exfoliation or desquamation of the epithelial cells; this results in the appearance of a peculiar mottled design. This change is commonly associated with or follows bedewing of the epithelium and resembles the picture observed after the instillation of cocaine solutions. Depending on the disease process, epithelial edema may be evanescent or permanent. Its presence only rarely predisposes to erosions, ulcer formation, and to deeper changes in Bowman's zone.

MINUTE EPITHELIAL AND SUBEPITHELIAL CHANGES

In addition to edema of the epithelium, certain delicate changes are seen in these layers, some of which are described under the heading of degenerations and others under the inflammatory conditions. Their exact significance is still unknown (Fig. 164). Briefly enumerated they are: (1) fine punctate dots in the epithelial surface, which may be indiscriminately scattered or appear in chains; (2) intra-epithelial foci; (3) subepithelial lines; (4) fissures; (5) epithelial erosions; and (6) epithelial filaments (Fig. 220; Plate XXXII, fig. 2).

These minute epithelial changes may follow the collapse of vacuoles or they may be due to intracellular inclusions or intercellular coagula. In other instances, when breaks in epithelial continuity occur (herpetic keratitis), marginal thickening of the fissure walls with increased relucency may form whitish lines. Fine epithelial filaments may develop on the cornea. These are firmly attached at the base and have a knoblike free end, which stains with fluorescein. They are thought to be idiopathic but are known to occur after herpes, erosions, keratitis sicca, filamentary keratitis (page 470). Fluorescein is of assistance in localizing and examining some of these structures, although all are not stainable.

VASCULARIZATION OF THE CORNEA

The avascular cornea derives its nutrition chiefly from the pericorneal vessels. In inflammatory conditions additional blood supply is obtained by two mechanisms: (1) pericorneal congestion or injection of the normal limbal vessels, and (2) formation of new vessels, which enter and vascularize the cornea (neoformation). The appearance of corneal vascularization is a definite indication of the beginning of reparative processes. Johnson and Eckardt¹⁶⁰ in their studies of ariboflavinosis pointed out that "the proliferation of capillaries from the limbus into the cornea is easily explained, therefore, as an attempt by the cornea to overcome local asphyxia by bringing available oxygen into closer proximity with the cells of the cornea." The constant appearance of corneal vascularization in albino rats having ariboflavinosis suggests that the deficiency of this vitamin (riboflavin) affects the corneal oxidative system and stimulates compensatory vascularization. Vascularization may develop early in a disease following the initial changes; or it may be significantly delayed. Late development may explain the chronicity and difficulty of clearing observed in some cases.

There is considerable clinical evidence that obliteration of newly formed vessels in various forms of vascularized keratitis (e.g., by diathermic coagulation of tributaries at the limbus or by peritomy) has a salutary effect in hastening healing.¹⁴¹ It has been felt for some time that in the cornea, nature's method of repair by vascularization may lead to further complications, such as bringing added noxious substances (e.g., anaphylactic antigens), which may increase the inflammation (as seen in phlyctenular keratitis) or produce an increase in scar tissue. However, in certain corneal inflammatory processes, such as interstitial keratitis, regression does not occur until corneal vascularization has been established. Thus Gundersen, in discussing these differences in the effects of vascularization, stated that "it must be assumed either that corneal vessels have diametrically opposite functions under different conditions or that the interpretation of the function of blood vessels in one or the other concept is at fault."

PERICORNEAL INJECTION

Pericorneal injection results from engorgement of the normal pre-existent limbal vascular arcades. Within the biomicroscope, the limbal arcades, which are normally threadlike, are seen to be engorged and many potential channels which ordinarily are invisible have become filled with blood. These loops, which may exhibit a granular type of corpuscular circulation, extend slightly over the limbal margin. The neighboring conjunctival vessels also become congested; this results in the so-called pericorneal injection. When pericorneal injection is intense and involves the deeper vessels, it is called ciliary injection.

NEOFORMATION OF VESSELS

Later, actual invasion of the cornea by blood vessels may follow. This occurs in two planes, superficial and deep. Depending on the severity of the involvement, vessels may migrate from one level to another. It is easier for the vessels to invade the cornea in Bowman's zone and in the region of Descemet's membrane than in the middle layers of the dense stroma (Plate XXVI, figs. 5, 6).

Superficial Vessels. The superficial vessels are derived from the conjunctival and episcleral vascular system and proceed into the cornea under the epithelium, sometimes raising it but more often coursing slightly deeper, invading Bowman's zone. This process may destroy Bowman's membrane and allow the vessels to penetrate into the anterior stroma. The development of superficial blood vessels has been studied in great detail with the biomicroscope. Kreiker¹⁸⁰ found that the invasion of the vessels occurs by the formation of capillary loops, advancing toward the center of the cornea, and that anastomosis results when two loops meet. Ehlers⁷⁷ stated that the new vessels make their appearance by the formation of long slender buds, which, after meeting, form loops from which new buds may develop. However, these buds may give the impression of being an elongated loop, the sides of which are so closely approximated that macroscopically they appear as a single blind

vessel. Detailed study by retro-illumination and high magnification discloses that actual budding of single blind capillaries may occur. This point has been demonstrated by Graves,¹²⁸ who made exhaustive biomicroscopic studies of superficial corneal vascularization. New buds or advancing endothelial tubes, which he designated as "pilots," are formed and directed to areas where the inflammatory focus stimulates their advance. As the new channels develop, older ones, now superfluous, become obliterated. Graves¹²⁹ states that "it does not necessarily follow that pilots will function; they may develop and then before the circulation has followed in their initial lead, its activity may subside or be deflected in another direction." Corresponding to the normal limbal circulation (page 145) and to the general pattern of all capillary loops, these newly formed loops are composed of afferent and efferent portions. The afferent vessels are of smaller caliber and change into a wider efferent vessel of venous character at their crests. According to Swindle,²⁹¹ the capillary loops extend into the cornea by a stretching mechanism rather than by an actual proliferative process.

Superficial vascularization occurs in many inflammatory conditions of the cornea, particularly in those associated with loss of substance (ulcers), keratitis eczematosa, rosacea keratitis, keratitis fascicularis, and in pannus.

Pannus (Latin — "cloth") consists of a superficial vascularization of the cornea accompanied by granulation tissue (Plate XXVII, fig. 1). This process occurs between the epithelium and Bowman's membrane, with destruction of the latter in most cases.* Pannus is also associated with keratitis eczematosa, rosacea keratitis, and keratoconjunctivitis phlyctenulosa; it may occur in degenerating eyes following severe intra-ocular disease, especially glaucoma.

Deep Vascularization. Deep vascularization of the cornea may follow severe corneal lesions which originate in the superficial layers (phlyctenular keratitis, rosacea keratitis, and severe ulcers of the cornea). The vessels succeed in migrating into the deeper layers, owing to the destruction of Bowman's membrane and softening

* The trachomatous type is described in the chapter on the conjunctiva (page 210).

PLATE XXVII

FIG. 1. Pannus trachomatosa in direct and proximal illumination.

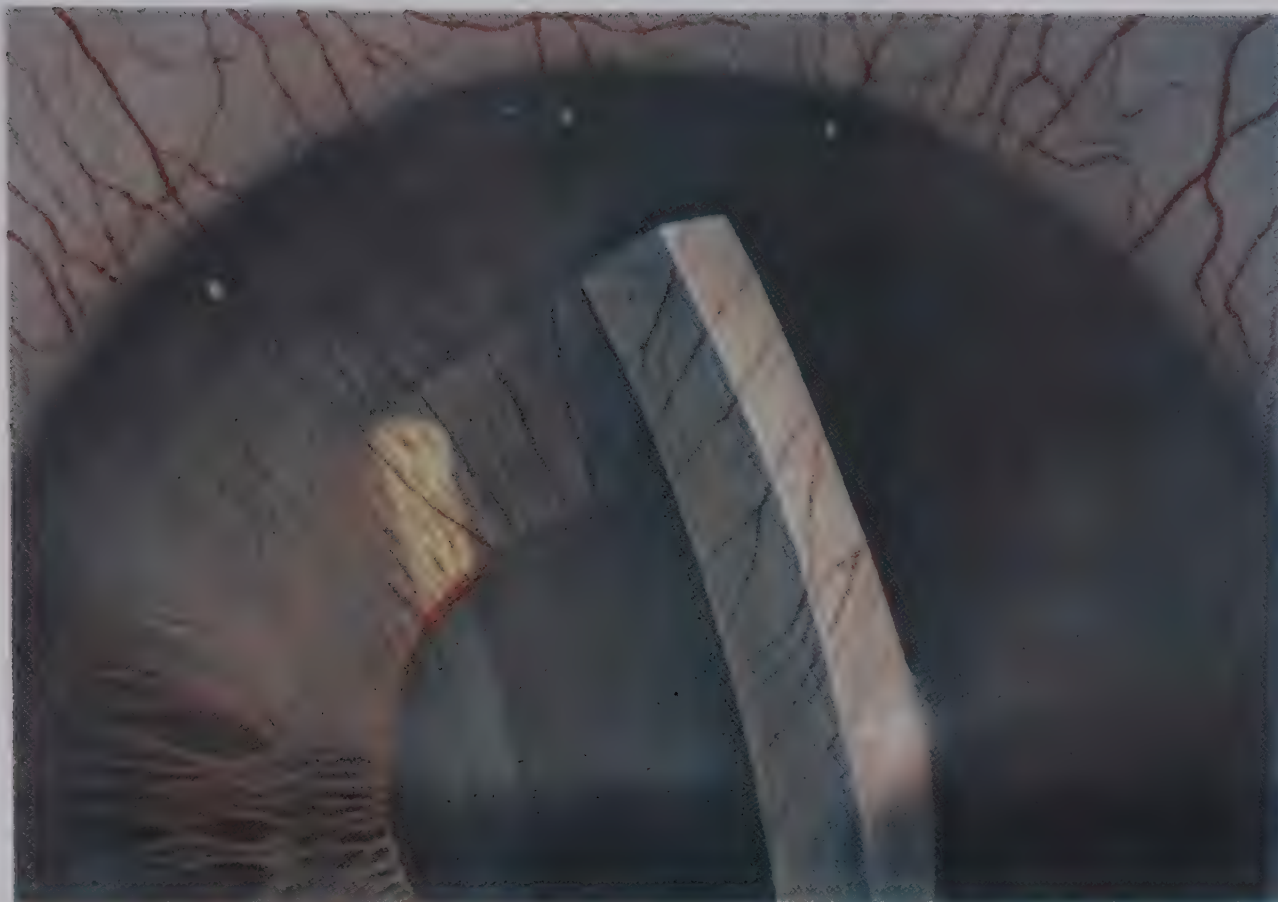
FIG. 2. Folds in Descemet's membrane seen by direct focal illumination and specular reflection; the dark tracts are outlined by two linear light reflexes. The folds are crossed by deep branching vessels.

FIG. 3. Folds and vascularization of the posterior corneal surface three weeks following cataract extraction (direct focal illumination and direct retro-illumination).

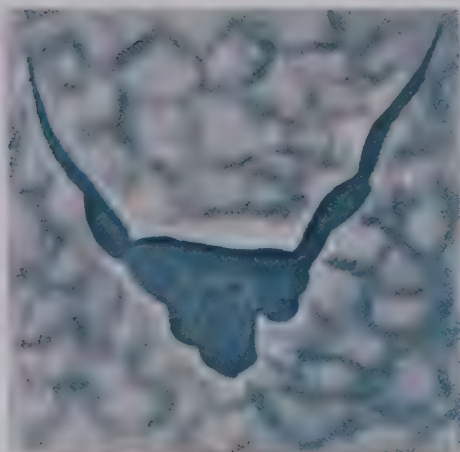
FIG. 4. Crocodile shagreen (Vogt).

FIG. 5. Folds in Descemet's membrane. 40 \times .

FIG. 6. Rupture of Bowman's membrane. Retro-illumination, 40 \times .



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of the corneal lamellae. This might be considered a secondary deep vascularization. Vascularization varies greatly, depending on the disease process. If the central portion of the cornea is affected,

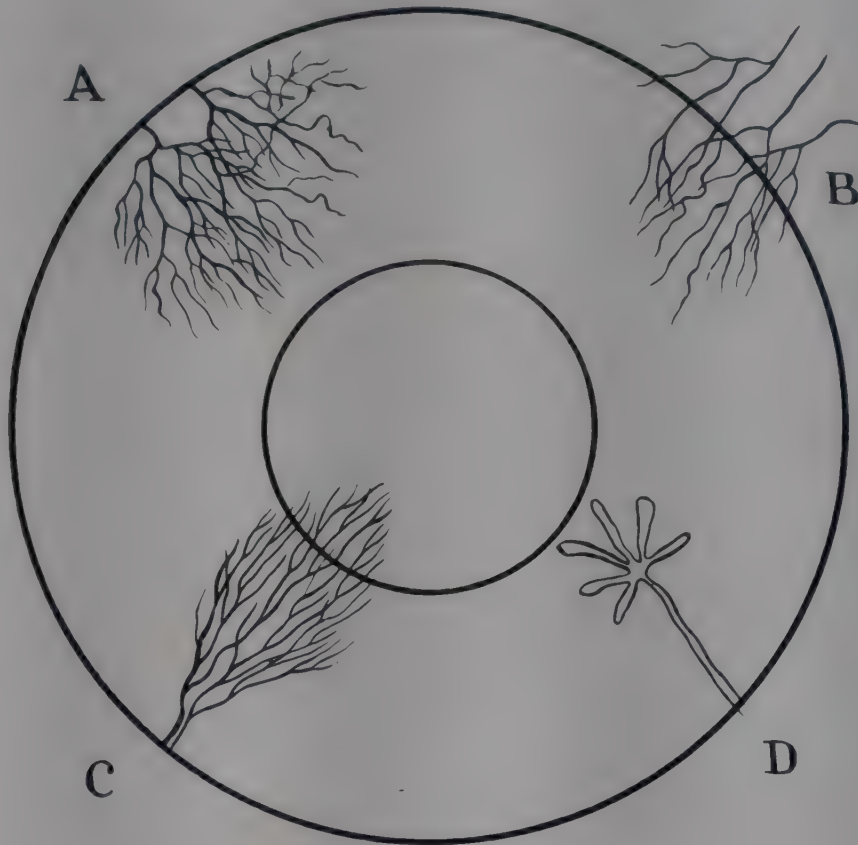


FIG. 197. Diagram of limbal neovascularization by retro-illumination. Composite of various types. A, Arborescent; B, terminal loop type; C, brush; D, umbel. (After Holmes-Spicer.)

vessels may penetrate from around the entire limbus. *Primarily*, the middle layers of the parenchyma are vascularized in such conditions as tuberculosis and disciform keratitis, and the deepest layers (Descemet's zone) in syphilitic interstitial keratitis, although no rigid rules can be made. In certain types of central involvement, for instance, keratitis profunda and interstitial keratitis, the appearance of corneal vascularization may be delayed. This may cause a persistent reaction. The vascular channels in primary deep vascularization originate from the anterior ciliary perforating vessels. In contrast to superficial vascularization, in which the vessels arborize, the course of the deep vessels, according to Spicer,²⁷⁹ may be classified into three types (Fig. 197): (1) terminal loops (e.g., marginal keratitis); (2) brush-form (parallel branches) seen in the early stages of interstitial keratitis (salmon patch); (3) umbral type, in which a large vessel extends into the cornea

and then terminates in a bud resembling a star. This type is seen in severe localized corneal conditions (e.g., rosacea keratitis and severe ulcers).

LOCALIZATION AND ADDED DETAILS CONCERNING NEOVASCULARIZATION

With the biomicroscope, it is a simple matter to localize the depth of vessels in the cornea. This can be accomplished (1) by the use of optic section and (2) by stereoscopic comparison with opacities of known depth. In most instances the depth of the vessel corresponds to the depth of the corneal lesions.

Corneal vessels are best seen by retro-illumination. By this method, even the smallest capillaries can be seen and identified. Large superficial vessels, such as those in pannus or phlyctenular disease, may have such a superficial situation that elevation of the epithelium occurs, causing the vessels to stand out in relief, giving rise to specular reflexes. In many cases of superficial inflammatory keratitides, smaller vessels are seen below the epithelium, invading Bowman's zone or even the superficial layers of the parenchyma. Such vessels may appear as whitish lines when examined in direct focal illumination but retro-illumination reveals their true nature. Deeper vessels which penetrate into the cornea in an arrow-like fashion tend to arborize around lesions. In cases of severe rosacea keratitis or phlyctenular keratitis, the presence of deeper invading vessels, associated with deep infiltrations, may lead the student to confuse these conditions with processes primarily interstitial, such as tuberculous keratitis or syphilitic parenchymatous keratitis. The deepest vessels, as seen in syphilitic interstitial keratitis, enter the cornea in Descemet's zone in the early stages; they are seen as a sector-like penetration of dull red parallel lines, surrounded by a reddish pink infiltration (salmon patch) (Plate XXXV, fig. 3). Later, finer branchings may follow the disposition of the infiltration and make their way into the middle and superficial parenchymal areas, depending on the extent of lamellar disintegration.

After the acute stage of interstitial keratitis has subsided, the

residual vessels form an extensive network overlying the sclerosed and relucet posterior corneal face. This appearance is typical of healed interstitial keratitis. In other conditions, there is usually a looser arrangement in which the vessels course irregularly through the various layers of the infiltrated parenchyma. In severe parenchymatous keratitis, the presence of engorged superficial vessels migrating over the cornea near the limbus, secondary to disturbances in Bowman's zone, leads to the development of a superficial corneal vascularization (epaulette). Superficial vessels may cast shadows, seen as dark lines, projected on the posterior face of the parallelepiped. Small superficial loops may also cause axial shadows. In other instances, deeper vessels, when seen in optic section, cause shadow lines to project back into the deeper part of the section. The vessels themselves, after penetrating the section (then becoming extrasectional) are seen in the reflected light from the deeper parts of the section as lines which are reddish or dark, depending on their size. As a rule once the cornea has been vascularized some evidence of vessels always remains. As the disease process resolves the vessels diminish in size and tend to become attenuated, but they never disappear completely. Attenuated vessels appear as delicate whitish lines which may be confused with nerve fibers in direct focal illumination; with strong retro-illumination, their true nature is revealed. Such vessels can be differentiated from nerves by the fact that the latter have a dichotomous branching and are generally invisible by retro-illumination; by this method of observation the vessels appear as faintly yellowish tubes, in which a granular circulation may frequently be seen. Visible capillary circulation may be present years after healing. Stroking the cornea or pericorneal regions can cause dilatation and even return of the granular circulation in apparently obliterated vessels.

NONSPECIFIC CHANGES IN BOWMAN'S MEMBRANE

In a sense, as in the case of other glass membranes (Descemet's, lens capsule and Bruch's membrane) of the eye, Bowman's membrane may act not only as a protective barrier guarding the deeper

corneal layers against the invasion of noxious substances but also physiologically as a selective filter for the purposes of maintaining corneal health. Loewenstein¹⁹⁷ states that "while the other tissues have to be nourished only, and moderate differences in the chemistry of the nourishing fluids would not be of great importance, the transparent tissues demand an exact constitution of the nourishing fluid in osmotic pressure, and special ions."

In order to observe and localize alterations occurring in this zone, it is necessary to resort to the use of the optic section, since with the parallelepiped this zone of discontinuity is not sharply outlined. Because of the lack of demarcation between Bowman's membrane and the subjacent corneal lamellae, it is impossible to know where one ends and the other begins. Consequently, this ill-defined region is aptly called the "Bowman zone" (Graves). Increased relucency in this zone is probably one of the most common pathologic alterations seen in corneal disease. It would appear that this zone effects a veritable superficial anatomic route for the entrance and extension of vessels and infiltrations from the periphery. The pathologic changes in Bowman's zone, as seen biomicroscopically, may be classified as follows: (a) opacities (resulting in granular, linear or diffuse increase in relucency and thickness); (b) honeycomb effect; (c) folds; (d) tears; and (e) vascularization.

OPACITIES

When Bowman's membrane is damaged it does not regenerate, but is replaced by connective tissue, which in optic section is manifested by an increase in relucency and frequently by an increase in thickness of this zone. Contraction may occur, producing simple concavities or even folds (page 411). Irregularities and loss of substance in this area are compensated for by an increase in epithelial thickness so that the actual surface of the cornea may not show contour defects or irregularities. Minute alterations in Bowman's zone such as granular excrescences or linear opacities of unknown origin (probably minor proliferation or destructive changes) are

frequently seen. These changes rarely raise the epithelium. All the aforementioned changes may be found in deep as well as in superficial corneal involvements, whether of inflammatory, degenerative, or traumatic origin. Increases in relucency of Bowman's zone have been noted even after long standing iridocyclitis, thus illustrating the fact that deep lesions of the eye may affect the superficial layers of the cornea.

HONEYCOMB EFFECT

Honeycomb effect (Graves) is caused by small excrescences, projecting from nebulae toward the epithelial surface at more or less regular intervals, the intervening areas being depressed. This gives the opacity a honeycombed or bosselated appearance, which is seen especially in herpetic scars, superficial dystrophies and in band-shaped keratitis.

FOLDS IN BOWMAN'S MEMBRANE

Folds in Bowman's membrane are relatively rare, and are seen in direct focal illumination as elevated undulated lines, having a double contour. They may be extremely irregular, varying greatly in size, and usually occur after severe inflammatory lesions or traumas (Fig. 208).

In most instances, folding of this membrane accompanies corneal scarring. Folds in Bowman's membrane may also occur, for example, after deep embedding of a foreign body, with perforating wounds, and in the cornea of shrunken globes. The close and indistinguishable attachment of this membrane to the underlying stroma in all probability would result under stress in tearing rather than folding. Butler⁴² described a case of old central keratitis, in which there was a folding in the form of radiating lines in both Bowman's and Descemet's membranes. Two star-shaped figures resulted, one behind the other. In superficial linear keratitis¹⁵³ (page 459), the spontaneous appearance of whitish superficial lines has been attributed to the formation of folds of Bowman's membrane.



FIG. 198. Optic section of tears in Bowman's membrane.

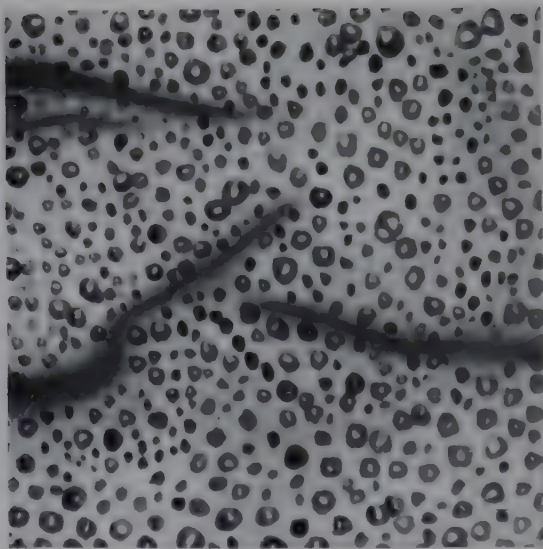


FIG. 199



FIG. 200

FIG. 199. Edema of epithelium and tears in Bowman's membrane in absolute glaucoma (retro-illumination).

FIG. 200. Tear in Bowman's membrane. Counterpuncture corneal wound resulting from corneal section with a Graefe cataract knife. The peculiar double-linear scars in Bowman's zone resemble a tear.

TEARS IN BOWMAN'S MEMBRANE

Tears in Bowman's membrane appear as irregular streaks which may be either parallel to each other or crossed (Fig. 198). The edges of the tears are usually not ragged. In fresh tears (Plate XXVII, fig. 6) there are clear intervening dark spaces between the reluctant edges of the lesions (Fig. 199). Later, owing to filling in by new fibroblastic tissue, the clear spaces disappear and dense superficial opaque bands may result. In the young tears may follow injuries or increased intra-ocular tension, especially in buphthalmos and in keratoconus, where they are seen as irregularly crossing white lines. Smaller, fine crossing lines have been reported following trephine operations. Fissures in Bowman's membrane may account for some of the reticulum in lattice dystrophy. Figure 200 shows a clear rent in Bowman's zone at the point of exit of the Graefe knife in a corneal section seen two weeks after cataract operation.

VASCULARIZATION

All superficial vascularization is subepithelial; therefore, it should properly be treated as one of the changes occurring in Bowman's zone; but because of its importance it has been discussed under the heading of superficial corneal vascularization (page 401). At first these vessels proceed between the epithelium and Bowman's layer but later, if there is destruction of Bowman's membrane, the vessels may go deeper. The migration of vessels is always accompanied by a slight increase of relucency in this membrane due to infiltrative, reactive or reparative processes.

ALTERATIONS IN THE STROMA BENEATH BOWMAN'S ZONE

These changes are: (1) edema and infiltration; (2) fissures, clefts and lines; (3) changes in thickness; (4) vascularization; and (5) scar formation.

Changes in the parenchyma are characterized by the appearance of edema and infiltration. In the beginning it is not always possible

even with the biomicroscope to differentiate between them. Parenchymal edema usually begins as a circumscribed or sector-like haziness (Fig. 225). In both instances the biomicroscope shows a diffuse haze, resulting from an increase in relucency, but infiltrations tend to assume a more or less granular or dotlike appearance (Fig. 226). Also in both cases, there is an increase in corneal thickness, usually manifested by bulging of the posterior corneal face in the involved area. As a result of this swelling, irregular lines or even dark clefts (Fig. 226 A), resembling water-slits, may be observed when the reaction is most intense. In keratoconus, systems of short parallel lines or interlamellar striae are found in the stroma; these are known as tension lines (Plate XXI, figs. 1, 3) and are probably due to fissures in the stroma. In addition, invading blood vessels are surrounded by faint zones of increased relucency. In the acute or progressive stages of a process, edema of the overlying epithelium may obscure delicate changes in the stroma. Severe reaction of the endothelial face (edema, precipitates, folds) tends to interfere with observation of the deeper corneal layers by retro-illumination.

Severe corneal edema is generally associated with folding of Descemet's membrane. Centrally, ringlike formations may result from marked swelling of the deep corneal face (keratitis centralis annularis of Vossius). As already indicated, there may be some difficulty in discriminating between edema and infiltration in their early stages. Biomicroscopically, it is impossible to see individual cellular infiltrates, but cellular aggregates which manifest themselves by a still further increase in relucency and finally by the development of definite opacification are easy to discern.

At first, infiltrates may appear as small whitish dots, which later become conglomerated. Sometimes, they assume a linear form resembling short white lines. In interstitial keratitis, the opacification in its earliest manifestation is seen in Descemet's zone. In other conditions, the infiltrates may begin in the more anterior portions of the parenchyma as dense, relucet, granular areas in the periphery, which shade off toward the center. When intense, such opacities

have a porcelain-like appearance, which may at times involve the entire corneal thickness (tuberculous keratitis). It is conceivable that when infiltration is dense, normal reparative processes (e.g., vascularization) are unable to effect complete resolution and, therefore, fibroblastic changes with resulting scars will occur, giving rise to permanent opacification. This is particularly true when destruction of the corneal lamellae has taken place. Laminated scars or opacities, so-called because in optic section linear-appearing alternating bands of opacity and relatively clear intervening layers of corneal parenchyma are seen, are found in a variety of conditions. If edema and infiltration are slight, they usually disappear. When the infiltration is caused by a pyogenic infection, abscess formation with loss of substance occurs, followed by scarring and finally thinning of the cornea.

Deep permanent opacities may show clearing stripes (Fuchs). Vascularization of the stroma tends to be permanent, but many of the vessels may become attenuated and in optic section appear as whitish lines.

ALTERATIONS IN DESCMET'S ZONE

The posterior corneal face is designated as Descemet's zone, because in optic section it is not possible to distinguish easily between Descemet's membrane and its underlying endothelium. However, in contrast to the epithelium, the endothelium can be seen in the zone of specular reflection. The changes which occur in this zone are: (a) edema; (b) hyaline striae and excrescences; (c) folds; (d) tears; (e) vascularization; and (f) precipitates.

EDEMA OF THE ENDOTHELIUM (DEWLIKE CHANGES)

Whether or not the endothelium is affected by edema which can be discerned by means of the biomicroscope is debatable. Vogt states that in acute iritis, he observed dewlike changes in the endothelium. In direct focal illumination the posterior corneal surface appeared to be covered by white dots; by retro-illumination these dots resembled

a mosaic of delicate, uniformly-sized dark droplets (Plate XXVI, fig. 2). Koeppe believes that these structures are transparent leukocytes rather than droplets resulting from endothelial edema. However, it may be stated that the single-layered endothelium does not present the typical picture of bedewing, so common to the surface epithelium. But when it does occur, it is seen more in the form of regular isolated droplets rather than as a dense carpet of closely-placed, irregular vacuoles. Keratic precipitates (fibrin, cells, and pigment) are generally interspersed between endothelial droplets or vacuoles. A similar droplike appearance may be observed upon the endothelium following opening of the anterior chamber (traumatic or surgical). In these instances, the appearance of bedewing is probably caused by irregularities or buckling of the endothelial layer. When edema of the epithelium and endothelium occurs coincidentally, differential diagnosis, under retro-illumination employing alternate superficial and deep focusing of the microscope, is not difficult (Plate XXV, figs. 3, 4). The endothelium reveals defects (dark spots), when observed by specular reflection, similar to the surface changes occurring in epithelial edema, when viewed by the same method.

HYALINE STRIAE AND EXCRESCENCES

Hyaline striae and excrescences are small lines, which in optic section appear as minute elevations on the posterior corneal face, protruding into the anterior chamber (Fig. 202). They are frequently seen in interstitial keratitis and are possibly caused by thickening of Descemet's membrane (Stähli), by localized proliferated endothelium (Graves), or by hyalinization of exudates deposited on folds. In addition to hyaline striae, small rounded protuberances of Descemet's membrane which project toward the anterior chamber can be seen by specular reflection (Plate XXVIII, fig. 1). These formations appear as dark, rounded areas in the endothelial mosaic and are similar to the Hassall-Henle bodies and to endothelial changes in guttata. Rapid oscillation of the beam or variation in the angle of observation causes a reflex from the center of these areas indicating

that some degree of concavity is present (crater-form). Vogt estimated the diameter of these concave protuberances as being from 20 to 100 microns. They are likely to appear after an acute inflam-

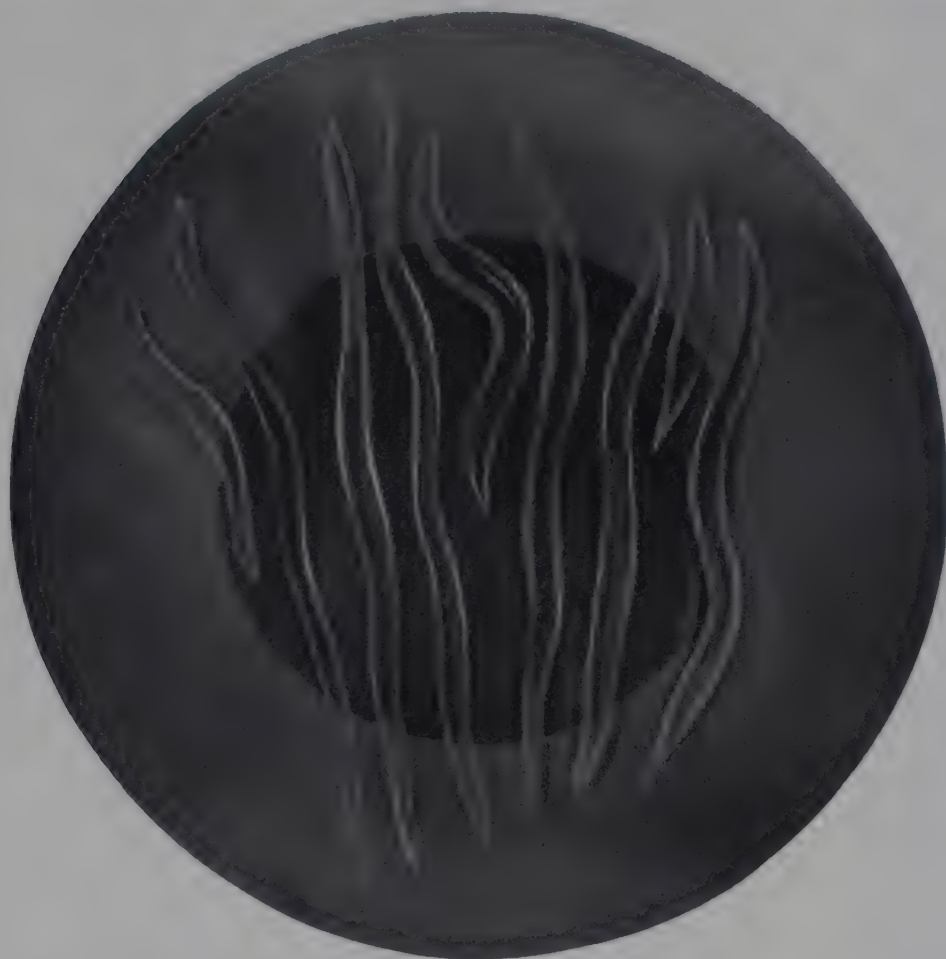


FIG. 201. Folds in Descemet's membrane in a case of interstitial keratitis.

mation has subsided. These formations are always larger than individual endothelial cells, appearing like dark craters in the luminous mosaic of specular reflection.

FOLDS IN DESCOMET'S MEMBRANE

The formation of folds in Descemet's membrane is so common that it must be considered as one of the chief ways in which this membrane reacts to insult (Fig. 201). Folds occur in numerous inflammatory, traumatic, and degenerative corneal conditions, whether superficial or deep; for instance, in interstitial keratitis, herpetic keratitis, keratitis disciformis, superficial corneal degenerations, and ulcers, as well as in severe involvements of the anterior segment (e.g., iritis). The formation of folds in the elastic Descemet's membrane undoubtedly results from corneal edema. Sudden lowering

PLATE XXVIII

FIG. 1. Shadow lines (folds) and protuberances in Descemet's membrane, in a case of quiescent interstitial keratitis. Direct focal illumination and specular reflection.

FIG. 2. Hyaline striae on posterior corneal surface. Retro-illumination. 40 \times .

FIG. 3. A case of epithelial vesicles and rupture in Descemet's membrane following trauma (direct focal illumination).

FIG. 4. Same case (Fig. 3) in optic section illustrating clear vesicles and effects on posterior face due to ruptures.

FIG. 5. Rupture in Descemet's membrane. Diffuse illumination.

FIG. 6. Rupture in Descemet's membrane (same as seen in Fig. 5) by sclerotic scatter and proximal illumination (high power). 60 \times . Note the curling edges of the tear.

FIG. 7. The rupture of Descemet's membrane. Direct focal illumination.



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of intra-ocular tension predisposes to the formation of folds (e.g., striate keratitis following cataract extraction). Folds are also commonly seen in atrophic globes with corneal shrinkage.

The biomicroscope demonstrates the frequency with which folds in Descemet's membrane are seen in severe keratitis. Diffuse illumination (prefocal or postfocal) usually reveals the entire extent and configuration of a well-pronounced fold. The smallest folds are always revealed by direct focal illumination and are characterized by the presence of two parallel linear luminous reflexes on each side of the fold, the ends of which converge and fuse (Plate XXVII, fig. 5). Between the linear reflexes a dark tract is seen, representing the part of the fold which does not reflect (Plate XXVII, fig. 2). Folds may occur in parallel groups; they may be branched or irregular in contour. In addition, there may be criss-crossing or interlacing of the folds and shadow lines (Plate XXVIII, fig. 1). Because these reflex lines are reflections from curved surfaces, a paralactic shifting in their position can be caused by varying the angles of illumination or observation. Vogt³²⁶ showed that these linear reflexes "behave similarly to those produced artificially on reflecting wave-like cylindrical surfaces. The width of the dark intervals between the reflexes will vary also depending on the angle of incidence of illumination and of observation. Although best seen in direct focal illumination, when they are deep they can be seen as sinuous bands in retro-illumination." The dark bands (shadow lines) which are seen crossing the endothelial mosaic in specular reflection result from the portion of the fold which does not reflect light (unilluminated portion).

The presence of double reflexes distinguishes folds from other corneal structures such as nerve filaments, vessels, or tears. Moreover, nerve filaments are finer and have dichotomous branches, while the true nature of vessels (circulation and color) is revealed by retro-illumination. Following cataract operation, folds may be so prominent that they may be seen by the unaided eye, in the form of numerous vertical or irregularly crossed parallel white lines (striate keratitis) (Plate XXVII, fig. 3). Although the lines may be

arranged radially, in most instances they form a criss-cross, irregular pattern. When deep scars are present, for example, following perforation, the folds may radiate from the scar. The deposition of exudates on folds in inflammatory conditions (e.g., herpes, tuberculous iritis, and interstitial keratitis) may result in the formation of hyaline striae on the posterior corneal surface. Whether or not these hyaline networks (*Glasleiten*) are caused by inflammatory exudates, or by the secretion of a new glass membrane by the endothelium, is unknown. It is possible that following severe edema and folding, the endothelium becomes detached and then secretes hyaline material, which is deposited on threadlike fibrinous anterior chamber exudates (Fig. 202). Vogt described clear circular glasslike deposits in healed cases of chronic iridocyclitis, surrounded by concentric or crescentic luminous halos, and resembling a gibbous moon. At times, he was able to discern endothelium on these deposits; and attributed the glossiness either to this covering or more likely to the wartlike prominences of Descemet's membrane.

TEARS IN DESCOMET'S MEMBRANE

Tears in Descemet's membrane may occur in conditions in which the cornea is subjected to sudden violent trauma (birth injuries or concussion) or to prolonged traction or distention (e.g., keratoconus and buphthalmos), or to surgical incision. They are most marked in cases of buphthalmos, in which they are seen as rather wide double-spaced sinuous lines with lighter hazy edges. These opaque areas, bordering the dark intervening spaces, assume the form of bands (Haab's band opacity).

Because of curling or elevation of the edges of the tear, small projections of tissue may extend into the anterior chamber (Plate XXVIII, figs. 3, 4, 5, 6, 7). Tears vary greatly in size; in keratoconus they may be small, while in buphthalmos they frequently range from 0.5 to 2 mm. in width (Figs. 157, 158). By means of specular reflection, it may be seen that the endothelium may be defective or even absent within the tear. Following trauma, Descemet's membrane may become separated from the subjacent stroma and, if



A



B

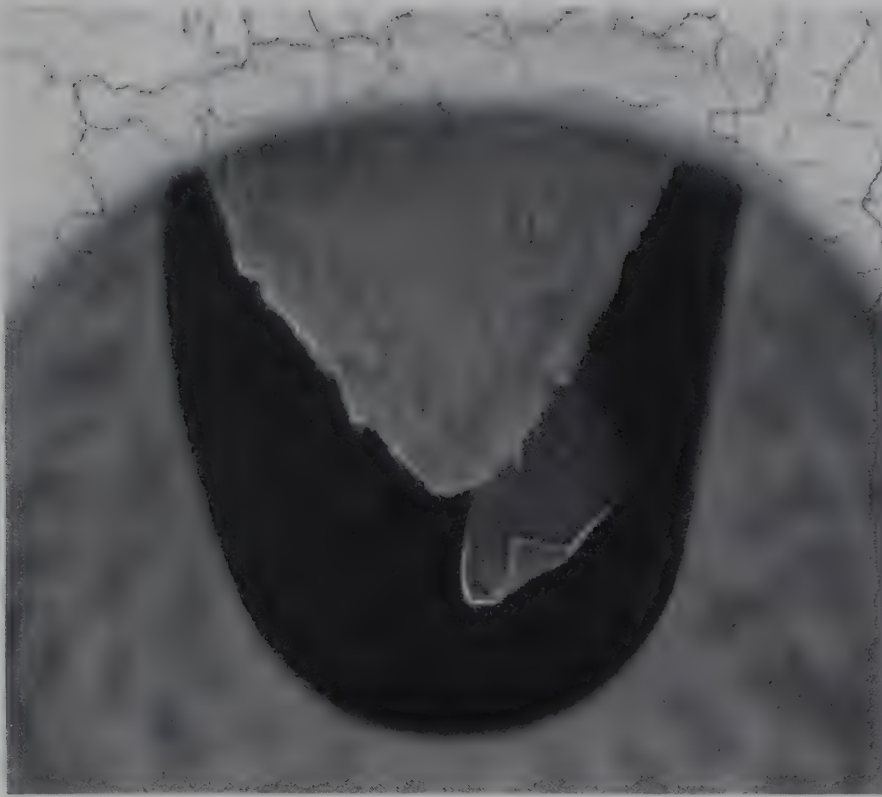
FIG. 202. Hyaline striae (*Glasleiten*) in (A) diffuse illumination and (B) optic section in interstitial keratitis. In A some of the parenchymal haze has been omitted in order to show the lesions more clearly.

it is attached at both ends, it appears as a chordlike hyaline band in the anterior chamber (anterior chamber, birth traumas, page 578), or as a sickle-like free strand (falciform rupture) if attached at only one extremity. The free end becomes bulbous or coiled up. The adherent end may spread out fanlike in singular or multiple attachments. Pigment granules may be deposited on these bands.

The free side-edges of a detached band, which may curl anteriorly in scroll-like fashion, are conspicuous because of brilliant light reflexes from the edges, even though they are translucent or transparent. This lesion may develop after certain types of operations. As a result of the forward spiral curling of the torn edges of Descemet's membrane, the new hyaline membrane in the rent fuses with the coiled edges on the convex surface and reduplication occurs, giving the appearance of several layers; such regeneration may also lead to the formation of a retrocorneal cyst.²³ When making a keratome incision in a glaucomatous eye with a shallow anterior chamber, splitting of the corneal stroma in the region of Descemet's membrane may occur; and a delicate sector-shaped deep opacity may develop, which remains permanently. Histologically, the endothelium regenerates over the exposed stroma and usually secretes a new hyaline membrane, which fuses with the old (Fig. 203).

If the cornea is examined immediately after an injury (e.g., birth injury after forceps delivery), an opacification of the cornea is seen. This is partially caused by the imbibition of aqueous fluid into the deep stroma through the rent in Descemet's membrane (corneal edema, page 414); when examined in optic section, the cornea appears thicker with a bulging posterior boundary. Eventually, the opacity may resolve partially or completely, in some cases leaving permanent bands of Descemet's membrane in the anterior chamber, bridging over the old gap, which has become filled through the development of new hyaline substance (Plate XXXVIII, fig. 5). The corneal stroma may revert to its previous transparency and thickness. Alternatively, a permanent opacity may remain owing to the formation of scar tissue.

A



B

FIG. 203. Detachment and opacification of Descemet's membrane following improper keratome incision. A. In diffuse illumination. B. In optic section.

KERATIC PRECIPITATES

Besides the presence of physiologic precipitates (lymphocytes and pigment) inflammatory states of the uvea are always characterized by the precipitation of cellular and fibrinous exudates on the posterior corneal surface. The impression that precipitates on the posterior corneal surface are caused by an inflammation of Descemet's membrane per se (descemetitis) was corrected by Fuchs.¹⁰³ He showed that such precipitates result from the deposition of exudative material on the posterior corneal surface, thrown out into the aqueous from the uveal tract. Therefore, Parsons²³² appropriately changed the meaning of the abbreviation "K.P.," formerly used to designate "keratitis punctata,"²⁷² to signify "keratic precipitates."

Koepe, who studied inflammatory keratic precipitates in detail, ascribed pathognomonic significance in some instances to the shape, size, or appearance of certain deposits. These findings have not been substantiated and most ophthalmologists now believe that no specific etiologic conclusions can be drawn from the character of the deposits in inflammation. However, histologic study has demonstrated that in the chronic granulomatous inflammations (tuberculosis, syphilis, and sympathetic ophthalmitis) there is a tendency for the deposits on the posterior corneal surface to assume large size (often visible to the unaided eye) and to have a waxy-yellow color (mutton fat). These deposits are comprised of plasma cells and large mononuclear phagocytes of various types. These cells have a greater tendency toward agglutination and adhesiveness than do ordinary lymphocytes and polymorphonuclear leukocytes.

In contrast to this, the polymorphonuclear leukocytes and lymphocytes seen in nonspecific inflammations have less tendency to agglutinate when suspended in the aqueous. This agrees with the clinical findings that the keratic precipitates are extremely fine and transient in such cases. In iridocyclitis associated with hypopyon, a carpet of more or less discrete white dots may be found on the posterior corneal surface above the hypopyon. The precipitates con-

sist of lymphocytes and it may be that the polymorphonuclear leukocytes gravitate down to form the hypopyon in the depths of the anterior chamber.

Except for rare deposits of foreign bodies, lenticular debris or exfoliated capsule, and physiologic deposits, keratic precipitates result from inflammation of the uveal tract. The presence of inflammatory products in the anterior chamber is revealed by increase in relucency of the aqueous, as shown by the flare of the beam (increased Tyndall phenomenon) (page 559). In the flare, cellular elements of various sizes are either still or move in a thermal convection current under the influence of gravity. The narrow beam or cylindric pencil aids in observing a positive Tyndall effect in the anterior chamber. Exudation of inflammatory products into the aqueous ultimately results in their precipitation on the posterior surface of the cornea. This is governed by the thermal convection currents of the aqueous fluid in the anterior chamber, as well as by ocular movements, the viscosity of the fluid, the adhesiveness of the precipitates, the effect of gravity, and the condition of the endothelium itself (anterior chamber, page 565).

Morphology. Deposits which occur in inflammatory processes, are composed of combinations of formed and unformed elements. These deposits may be pigmented or nonpigmented. In either case, keratic precipitates may distribute themselves in a variety of forms over the posterior corneal surface as follows:

1. Scattered dustlike deposits composed of either individual cells, pigment granules, or dots of cellular debris (Plate XXIX, fig. 1).
2. Small clumps and larger disks composed of aggregates of cells or debris (Plate XXIX, figs. 2, 3, 4).
3. Droplets, appearing as small, clear vacuolar structures considered by Vogt to be endothelial cell edema and by Koeppe to be individual white cells (Fig. 204) (see Turk's line).
4. Strings composed of cellular deposits or debris arranged in chains or lines. In some instances geometric designs may appear. These result from interconnection of deposits by lines of fibrinous coagulum (Spicer's star-ring type) (Fig. 205).



FIG. 204. Droplike deposits on the posterior corneal surface.

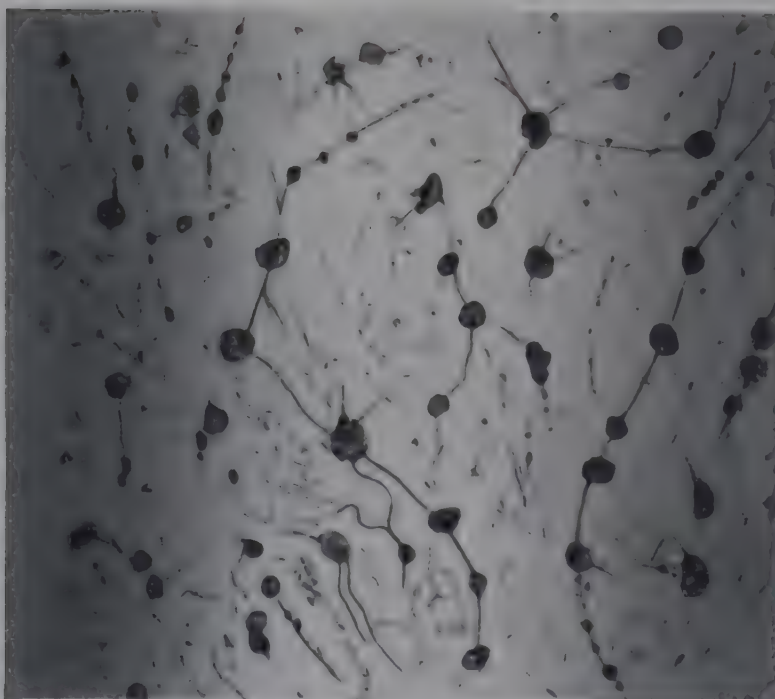


FIG. 205. Strings composed of cellular deposits and debris arranged in chains and starlike clusters on the posterior corneal surface. (Spicer star-ring type.)

5. Stellate deposits (arranged in the form of stars) (Plate XXIX, figs. 5, 6, 7).

6. Fibrin may be deposited in the form of sheets or plaques (Plate XXX, fig. 1).

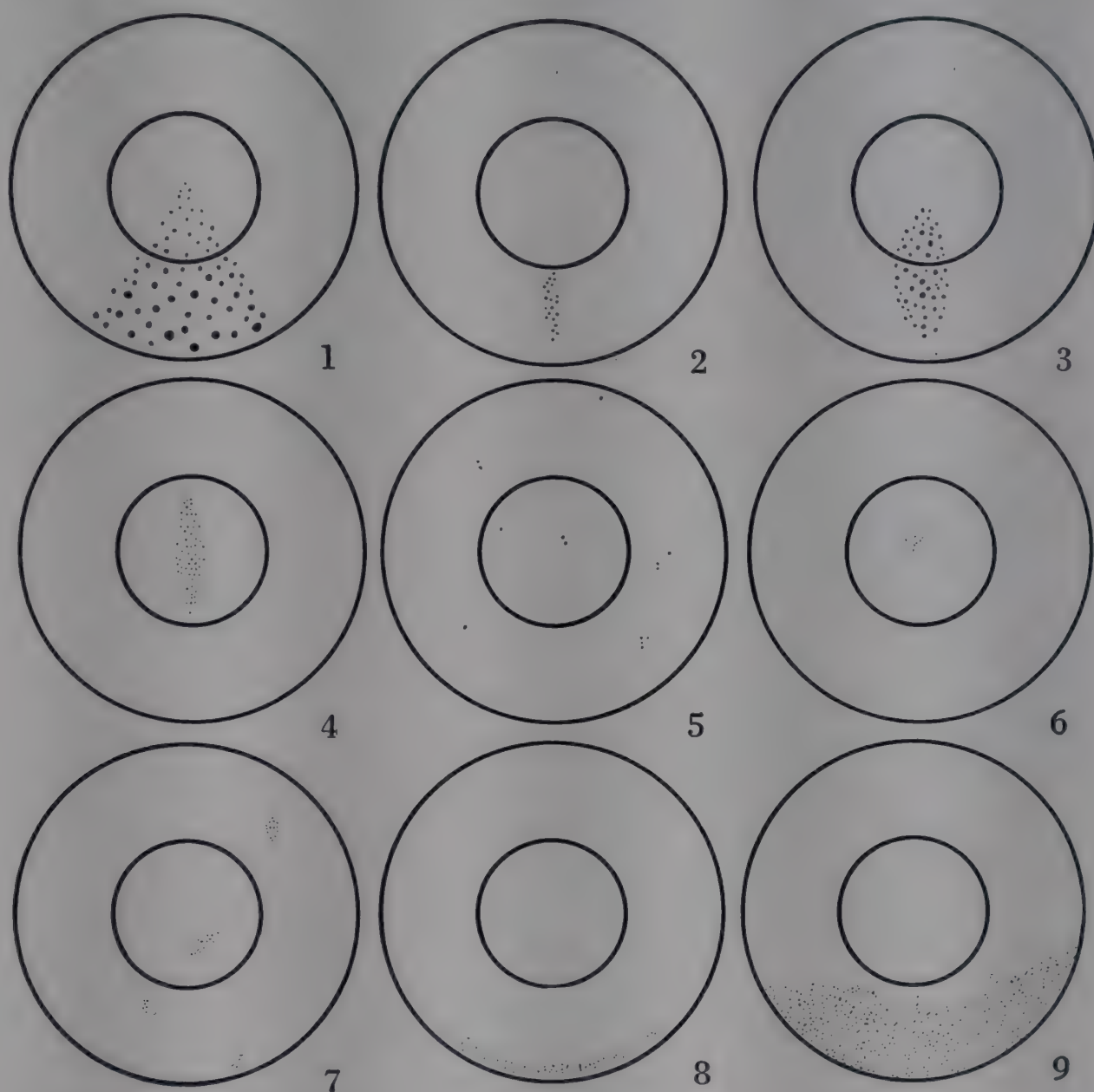


FIG. 206. Distribution of keratic precipitates. (1) Classical triangular (inverted V); (2) linear Ehrlich-Turk line; (3) and (4) fusiform (Krukenberg's spindle); (5) diffuse (disseminated over entire cornea); (6) localized centrally; (7) localized irregularly; (8) localized peripherally; (9) massive consisting of sheets or plaques.

Distribution of keratic precipitates may be classified as: (1) classical triangular (inverted V); (2) linear (Ehrlich-Turk line); (3) fusiform (Krukenberg's spindle); (4) diffuse (disseminated over the entire cornea); (5) localized (central, irregular, or peripheral); and (6) massive (gelatinous), consisting of sheets, plaques, gelatinous masses (Fig. 206).

PLATE XXIX

FIG. 1. Scattered dust deposits, individual cells, pigment granules and debris by retro-illumination.

FIG. 2. Small clump deposits on the posterior corneal surface.

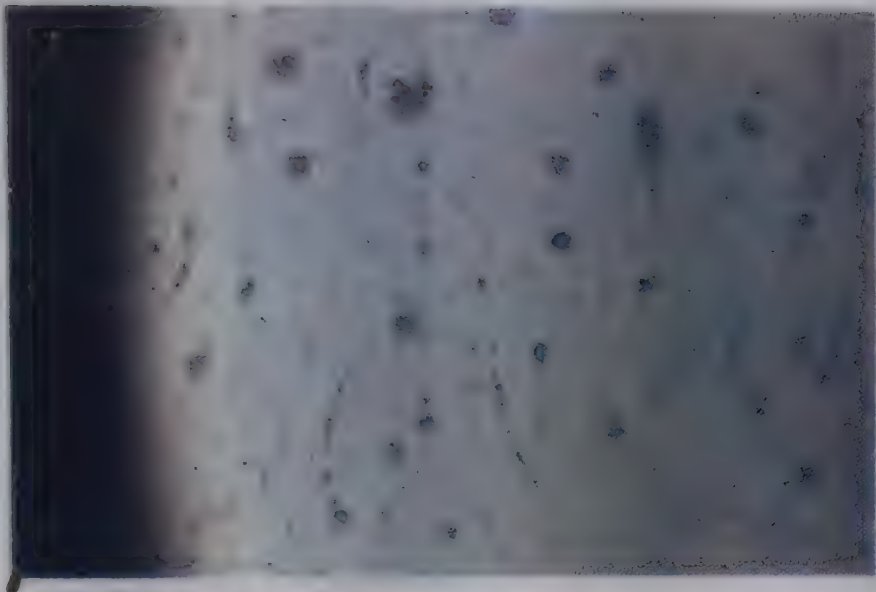
FIG. 3. Discoid keratic precipitates in direct retro-illumination.

FIG. 4. Keratic precipitates; discoid mutton fat type (high power) in direct focal illumination.

FIG. 5. Large stellate keratic precipitates. Direct retro-illumination.

FIG. 6. Large stellate keratic precipitates in direct focal illumination. (Same case as in Fig. 5.)

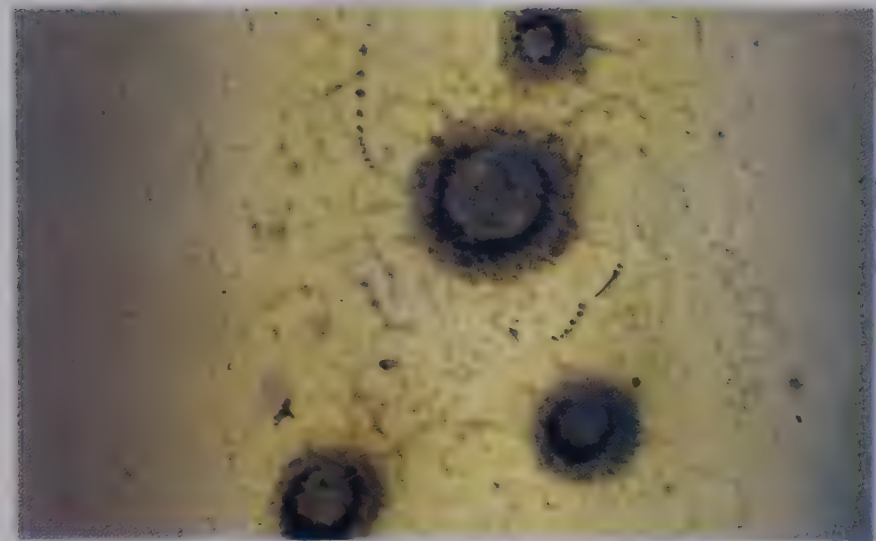
FIG. 7. Large stellate keratic precipitates by indirect retro-illumination. (Same case as in Fig. 5.)



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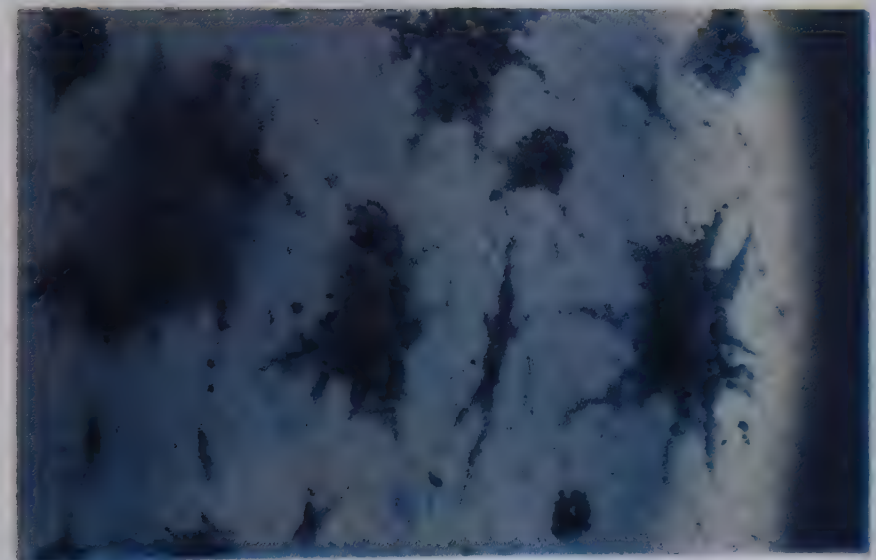
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As deposition and distribution of keratic precipitates are governed largely by physical forces (thermal convection currents and gravity) and the condition of the endothelium, local variations of these factors produce various arrangements of the deposits. Depending on the severity or the stage of the disease process, the distribution and arrangement may change from one type to another. The usual disposition of keratic precipitates in iridocyclitis is the classical inverted V or triangular formation on the lower part of the cornea. The size of the clumps tends to increase toward the base of the triangle. Ordinarily, the largest clumps occur in cases of cyclitis. The designation "mutton fat" has been given to certain deposits, especially those occurring in granulomatous cyclitides (tuberculous or syphilitic or associated with sympathetic ophthalmitis) because of their large size and grayish greasy appearance.

Deposits along vertical lines, single or multiple, occur either physiologically, as in the Ehrlich-Turk line, or in low-grade inflammations of the uveal tract. Fusiform depositions, likewise, appear in the questionable congenital spindle of Krukenberg and more rarely in inflammations.

Another type occurs in the form of a diffuse punctate dissemination over the entire posterior corneal surface. Although this type of dissemination has been found in low-grade chronic uveitis (e.g., heterochromic cyclitis), it may also occur in more acute lesions or in those in which the deposits consist only of pigment granules.

Precipitation may occur in localized areas on any part of the posterior corneal surface. The precipitates may assume a disklike arrangement in the pupillary zone of the cornea or, peripherally, they may appear as a bowlike arc, concentric with the limbus. The latter arrangement is frequently seen in chronic iridocyclitis and may be a residue resulting from partial absorption of the upper part of the triangular form. Irregularly disposed aggregates may occur directly behind a localized area of intracorneal inflammation (e.g., herpetic keratitis). As a rule during the early stages (even in the granulomatous uveitides) the deposits, whether localized or diffuse, occur

as a carpet of individual cellular elements. It is only as the disease progresses that aggregation into clumps or disks is seen.

Massive sheets or plaques of plastic fibrinous exudate, in which cellular elements may be admixed, are seen in cases associated with severe exudative uveal inflammation (e.g., gonorrheal iritis, and interstitial keratitis) (Fig. 226 B). Such adherent exudates appear in a variety of changing forms and locations, depending on the progress of the disease (Plate XXX, fig. 1). Complete resolution may occur or organization of inflammatory products, accompanied by vascular invasion in some cases, may result. Destruction of the subjacent endothelium may cause a permanent, dense, relucet, deep opacity.

Massive semitransparent gelatinous exudation into the anterior chamber may become adherent both to the posterior corneal surface and to the iris. This type of exudate commonly occurs in the early stages of gonorrheal iritis; as a rule it tends to absorb, or leaves a fine fibrinous detritus on the posterior corneal surface.

Physiologic Deposits. Keratic precipitates may occur in apparently normal eyes. Two forms are known: a nonpigmented cellular form and a form which consists of pigment granules. The presence of individual leukocytes on the posterior corneal surface (Turk's line) has been noted in the normal eyes of the young (from 7 to 16 years of age)^{139, 202} (Plate XXX, figs. 2, 3). Guggenheim noted this in 50 per cent of her cases. The cells (varying from 10 to 30 in number) are grouped along a vertical column 0.1 mm. in width and from 0.4 to 0.8 mm. in height. This line is usually found in the lower central region of the cornea with its upper pole on a level with the lower pupillary border. It is important to bear in mind the existence of this physiologic droplet line in order to avoid diagnostic errors. In this connection it is significant that the physiologic line is inconstant in comparison with its inflammatory counterpart. In addition, physiologic deposits occupy part of the Ehrlich-Turk line, whereas pathologic deposits occupy the whole line and surrounding areas and there are concurrent changes in endothelium, stroma and epithelium. In doubtful cases, a second observation after a few days

will usually resolve any doubt as to the pathologic nature of the deposits.

Although the Turk line generally occurs singly, Vogt described cases in which multiple lines were found. He observed that at times the constituent cells exhibit slight ameboid movements. These cellular deposits may be distinguished from pathologic precipitates by the fact that they do not agglomerate. The cells can be seen by means of direct focal illumination as well as by retro-illumination. Koby noticed that by retro-illumination, the outlines of such cells are lighter than their centers. By retro-illumination they appear as clear drops; by direct focal illumination, as fine white dots. These cells are considered to be leukocytes which originally circulated in the anterior chamber, but the possibility of their being migrating tissue cells (histiocytes) should be kept in mind. A similar type of deposition has been noted by Guggenheim in one third of her patients with corneal foreign bodies.

Pigment Deposits. Pigment deposits on the posterior corneal surface may occur independently as dots or granules or in stellate forms or in a mixed form; the pigment may be adherent to leukocytic clumps or disks (Plate XXX, fig. 4) (Plate XXIX, figs. 4, 6). It is impossible to determine whether pigment granules derived from broken-down pigment cells originate in the pigment epithelial layer (posterior iris) or from melanophores on the anterior iridic surface. The stellate forms may represent migrating melanophores. Pigment granules deposited on the posterior corneal surface are an almost constant finding in the aged even when there is no evidence of actual uveal inflammation. This probably results from atrophic or degenerative changes, incident to the processes of senescence. When occurring in the young, the cause is either inflammatory, or it is congenital, frequently in association with other anomalies (e.g., persistent pupillary membrane). This type of pigment deposition is noted in highly myopic eyes and in megalocornea. Pigmented granules on the posterior corneal surface have been observed in diabetes. Pathologically, in inflammations of the uvea, pigment granules, probably melanin, are usually dark brown in color and are seen in blue eyes as well as in

PLATE XXX

FIG. 1. Plaques. Exudative membranes on the posterior corneal surface. Iridocyclitis. Direct focal and retro-illumination.

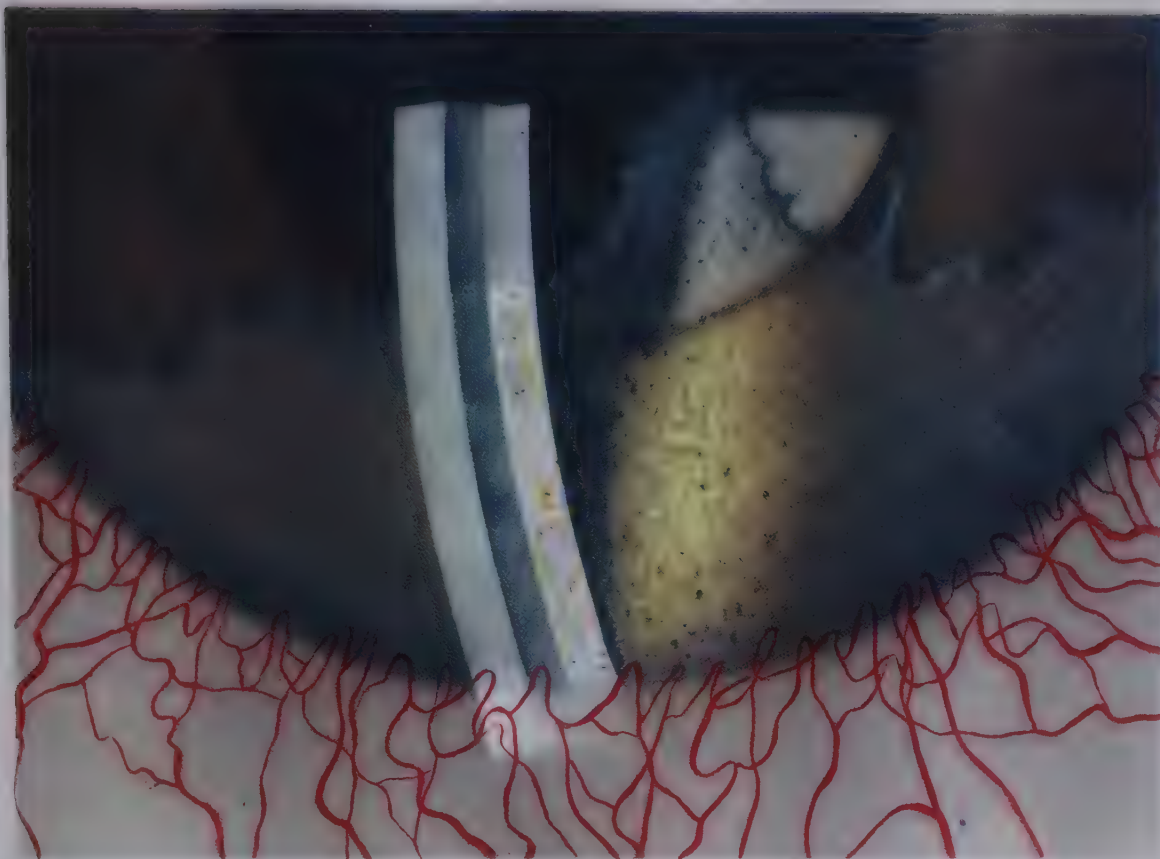
FIG. 2. Ehrlich-Turk line by retro-illumination.

FIG. 3. Ehrlich-Turk line in optic section. Direct focal illumination.

FIG. 4. Stellate and dustlike pigment deposits on the posterior corneal surface (retro-illumination).

FIG. 5. Large pigmented keratic precipitates showing reversed illumination effects. Interspersed are dustlike deposits and deep corneal vessels.

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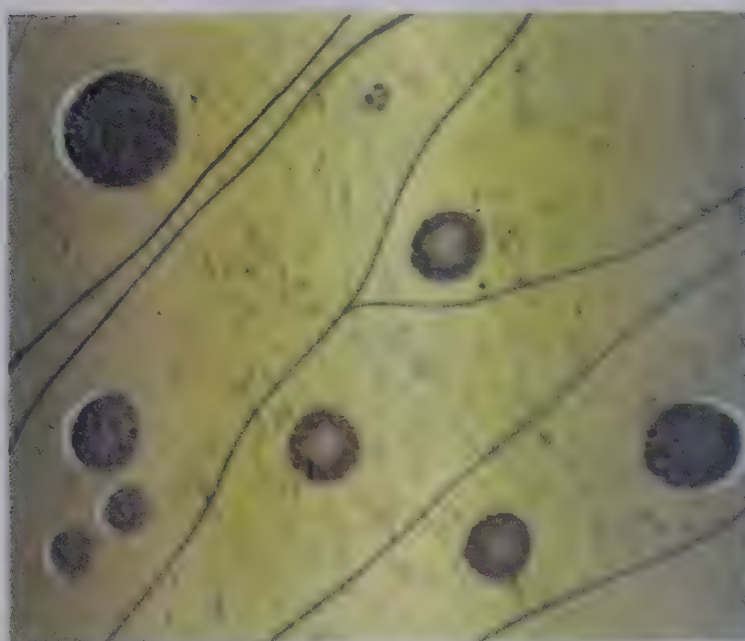
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brown; consequently, they must be considered to be derived from the retinal pigment layer. When fine, the granules may appear grayish. Although they may be found in the central area of the cornea in either linear or fusiform arrangement, their more common disposition is in the classical inferior triangular (\triangle) form.

After the absorption of keratitic precipitates their sites may become sprinkled by pigment of an amorphous powdery nature. It is not known whether the pigment was carried to these areas by the original leukocytic deposit or precipitated later from the aqueous as foci pigment. In some instances, the pigment deposit is not amorphous but may appear as starlike forms resembling those seen on the anterior lens capsule from the remains of the pupillary membrane. Old leukocytic deposits usually tend to become pigmented but the mere absence of pigment is not a proof that the deposits are fresh (Plate XXX, fig. 5).

Pigmentation, when dense and compact, may appear black. Under intense illumination, black-appearing pigment may seem to have a brownish red tint. This point can be demonstrated when examining the pigment excrescences on the pupillary border. When the light enters temporally, the temporal excrescences appear black while those on the nasal side appear reddish brown. This is due to the fact that more light is reflected to the observer's eye from the nasal than from the temporal side.

Blood-tinged precipitates are sometimes seen in hemorrhagic tuberculous iridocyclitis, in which the edges of the deposits may take on a reddish hue owing to the effect of gravity produced by the position of the eye.

In cases of hemorrhage into anterior chamber (hyphema), brick red deposits are sometimes seen on the posterior surface of the cornea. These are erythrocytic aggregates and may occur in isolated deposits or in plaque formation. Sometimes columns of small red dots, separated by clear areas, are arranged beneath larger clumps (Plate XXXI, figs. 1, 2). It seems as though blood cells drip down from these clumps toward the hyphema.

Biomicroscopic Technique and Optical Phenomena. The biomicroscope is of great importance in identifying and studying keratic precipitates. When large and dense, the presence of the deposits can be established by sclerotic scatter and proximal illumination. However, direct focal illumination with higher magnification ($20\times$ or more) is necessary in order to localize and properly to identify small deposits, consisting of detritus or individual cellular elements. Vogt showed that at times the most minute precipitates can be seen only in the zone of specular reflection of the posterior corneal surface. Here, they appear as small dark spots, which he compared to imperfections in the silvering of a mirror.

Moreover, by retro-illumination the exact location (in depth) of keratitic precipitates can be determined only by differential focusing of the microscope or by comparison with structures of known depth (e.g., epithelial changes). When using this form of illumination, the fact that the keratitic precipitates all lie in the same plane also aids in their localization. Dependent on whether direct or indirect retro-illumination is used and on the density of the deposits, they may exhibit certain special optical features. For example, in direct retro-illumination, when of solid density, a keratic precipitate may obstruct the reflected light completely and appear dark. If the precipitate is heavily pigmented, or if it consists of pigment alone, it is seen to be relatively dark against a bright background. However, if it is not completely obstructive, it may appear as a dark-rimmed circle with a light center. In indirect retro-illumination (Graves), scattering of the light by a precipitate (respersiveness) may make it appear relatively brighter than the dark background.

A clear vacuolar precipitate (edema of the endothelium or a clear leukocyte) exhibits refractile properties, as evinced by the distorted (minified or magnified) view of the junction between the light and dark areas of the background (pupillary border).

Solid precipitates demonstrate reversed illumination (Graves), when viewed against the dark background of indirect retro-illumination. The border of the precipitate which is farther away from the brightly illuminated iris is brighter than the nearer border (Fig. 78). On the other hand, vacuolar deposits exhibit unreversed illumina-

tion when examined in retro-illumination. The border of the precipitate nearer the light is brighter than the edge which is farther away. The study of precipitates by means of retro-illumination and the observation of the optical effects exerted by the deposit, reveals information concerning their structure, which cannot be obtained by direct focal illumination. For example, a deposit which seems to have a solid consistency by direct focal illumination, may have a granular structure by retro-illumination. In the same manner crenations of the edges of discoid keratic precipitates can be demonstrated. Such information assists in determining the age of a deposit. The solidity of a clump or disk is a much better indication of its age than the presence of pigment. Although fresh clumps may be non-pigmented, they tend to become darker and pigmented with age. However, this is not true in all cases, for it is well known that the white precipitates in heterochromic cyclitis rarely, if ever, become pigmented, even though this condition is associated with pigment destruction in the iris.

Differential Characteristics. In many cases of acute iritis, the precipitates are small, consisting of pigment dots and fibrin. Small fibrinous particles may appear as white lines in direct focal illumination and as dark lines by retro-illumination. However, in cyclitis, the large, solid mutton-fat type of deposit is commonly found. In the subacute or chronic granulomatous uveitides (tuberculous and sympathetic ophthalmitis), the deposits may assume discoid shapes. In mild cases, it is possible for such deposits to disappear in a few days; in chronic cases they may persist for months. As time goes on, these structures may change in shape and become pigmented. Absorption causes thinning and crenation of the edges, forming stellate patterns. Further absorption results in a fenestrated appearance due to separation of cells, the pigment remaining longest. Occasionally in discoid aggregates of moderate size, regression is attended by the formation of veil-like halos which may interconnect with adjacent halos leading to a netlike formation over the entire posterior corneal surface, the pigmented deposits standing out like chenille spots on a woman's veil.

Usually in inflammatory states of uveitis, the appearance of pre-

cipitates is preceded by an endothelial bedewing. This can be seen by retro-illumination and high power which reveals isolated droplets.

According to Vogt these droplets are the result of endothelial edema; Koeppe believed that they are mononuclear leukocytes. This initial change is followed by various forms of precipitates. Precipitates may excite reaction in the overlying corneal stroma (Plate XXVI, fig. 2). This is caused by the disturbed osmotic relationships, secondary to the damaged endothelium, or by the toxic effects exerted by the deposits, and is demonstrated by increased relucency in these regions. In severe cases the parenchyma overlying the precipitates becomes edematous and in direct focal illumination there is sudden widening of the optic section. The bulging is always posterior. Moreover, in many instances even the overlying epithelium may become edematous. Focusing the microscope backward and forward in retro-illumination alternately reveals the two planes of change. (Plate XXV, figs. 3, 4).

The exact mechanism of the formation of clumps or disks (mutton-fat deposits) is not entirely clear. It may be that in these instances the initial cellular deposit is highly toxic and leads to an increase in the local reaction and to a consequent increase in cellular deposition at this point. Following operation or injury, masses of lens material or capsular remains of varying size and shape may be attached to the posterior corneal surface, to the iris, or in the angle. In glaucoma capsulare, exfoliations from the lens capsule may be deposited upon the lower portions of the posterior corneal surface (appearing as small white flakes) or in the depths of the chamber angle.

INCREASED VISIBILITY OF CORNEAL NERVES

Many biomicroscopists have noted an increase in the visibility of the corneal nerve fibers. Some have attached an etiologic significance to this change. In practically all disturbances of the cornea, the nerves appear more prominent when examined in direct focal illumination. This may be explained by the fact that in pathologic

states changes in the index of refraction of the corneal gel may enhance the visibility of the nerve fibers. Duke-Elder⁶⁴ described a case of superficial vesicular keratitis, in which the nerve fibrils were

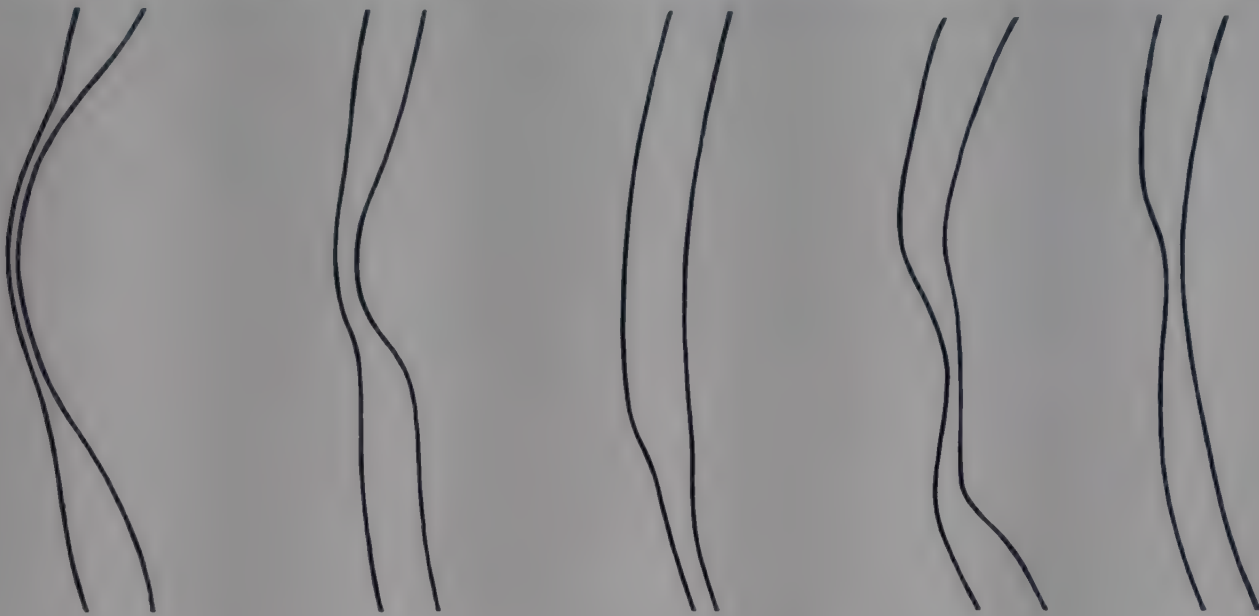


FIG. 207. Diagram illustrating variations in corneal thickness.

unusually prominent and terminated in bulbous extremities. The literature reveals many descriptions of corneal conditions, such as herpes, the dystrophies, leprosy, and keratoconus, in which prominent enlargement of the corneal nerves has been noted. In some of these cases the nerve filaments seemed to be directly connected with the lesion. In others, linear opacities seemed to be of neurogenic origin. In most cases, vital staining of the corneal nerves has revealed that the connection was only apparent. Consequently, in the present state of our knowledge, specific significance should not be attributed to all cases of increased visibility of the corneal nerves.

CHANGES IN CORNEAL THICKNESS

When examining a pathologic cornea by means of optic section, changes in corneal thickness are observed. Localized alterations caused sudden variations in the width of the beam, especially discernible when part of it lies over an unaffected area (Fig. 207). Although uniform changes in thickness involving the entire cornea are more difficult to estimate, the experienced biomicroscopist will easily detect such a change; if necessary, comparison with the thickness of a normal cornea will quickly enable the examiner to deter-

mine any variation from the normal. It should be noted that in the normal cornea, the periphery is a little thicker than the axial region. Koby recommends the use of his astigmatic beam to determine corneal thickness because the size of the elongated section corresponds favorably to the diameter of the cornea and variations in thickness are disclosed without the inconvenience of raising or lowering the usual smaller section. Lower magnifications which permit a larger field of view must be used. For more accurate determinations the micrometer ocular is available (page 120).

Increase in Corneal Thickness. This always occurs following parenchymal edema and infiltration and is marked in the areas in which these processes are most intense. The classical examples occur in interstitial keratitis and disciform keratitis, in which localized increases in thickness are usually observed as posterior bulgings. This type of bulging of the deeper part of the cornea apparently always accompanies cases which are associated with parenchymal edema or infiltration. It may be due to the fact that Descemet's membrane, because of its elasticity, stretches more readily than Bowman's membrane. Spontaneous or traumatic ruptures of Descemet's membrane result in imbibition of aqueous fluid into the cornea. This causes opacification and swelling of the parenchyma, which is seen as an increase in relucency and a bulging of the posterior corneal surface toward the anterior chamber. Certain superficial conditions, such as herpes, acne rosacea, and ulcers, produce endothelial disturbances and folding. Some local increase in corneal thickness may appear in such cases. Generalized uniform thickening and opacification of the entire cornea has been described as an early manifestation of interstitial keratitis (Koby).

Increase in corneal thickness caused by forward swelling of the anterior corneal layers may also occur. As a rule these are local in character. Superficial infiltrations, such as one seen in acute stages of acne rosacea, ulcers, pannus, lipin keratitis, and corneal dystrophies, produce a widening of the optic section in the areas in which they are most dense. The overlying epithelium when edematous and raised causes a similar change. In most cases the changes which produce an increase in corneal thickness are manifestations

of an early or acute process and consequently are subject to daily variations, eventually disappearing; or, depending on the amount of destruction, these changes may be followed by the opposite condition, the result of the reparative process, that is, corneal thinning. However, localized thickening may occur in corneae that have been previously thinned following the development of secondary degeneration in the form of hyaline, fatty or calcareous infiltration.

Thinning of the Cornea. Thinning of the cornea results from the loss of corneal substance following ulceration or from softening and atrophy. When lamellar material undergoes dissolution, it is replaced by scar tissue.* The dynamic effect of the intra-ocular tension is probably an added factor in that during the stage of softening the *vis a tergo* may cause further stretching and thinning out of the already weakened tissue. Thinning of the cornea, in contrast to thickening, is a permanent alteration and at times is progressive (keratoconus, ectasia). The classical example of corneal thinning is that associated with all corneal scars, even small superficial ones (Fig. 208). In optic section, such scars appear as a dense relucant opacity in Bowman's zone and are slightly depressed. The depression may or may not be filled out with epithelium, but in either case there is a distinct narrowing of the width of the section. Staining with fluorescein is of great value in studying delicate variations in thickening of the epithelium. When marked thinning of the cornea has occurred (keratoconus or marginal ectasia), the anterior and posterior corneal surfaces apparently almost touch one another. The anterior surface may be depressed but it is very rarely, if ever, that forward bulging of the posterior face occurs (keratoconus posticus). Marked thinning of the cornea is observed after healing in cases of rosacea keratitis and interstitial keratitis (chronic stage). However, in the former, in which there usually is considerably more loss of corneal substance caused by ulceration, the thinning is likely to be greater and more irregular than in the latter. Koby described a case of corneal thinning occurring years after a severe contusion of the globe. An unusual feature was that the cornea in the

* However, Salzer²⁶¹ showed that some regeneration of corneal tissue can occur provided infection does not intervene. In this case, keratoblasts (small spindle cells) elongate and form clear corneal lamellae.

thinned area was clear. He attributed the thinning to atrophy.

An example of extreme localized thinning is seen in cases in which following destruction of the entire parenchymal thickness,



FIG. 208. Optic section through corneal scar in a healed case of rosacea keratitis showing thinning of the cornea, folds in Bowman's and Descemet's membranes. Note superficial nebula in lower part of section.

Descemet's membrane bulges forward and forms a bleblike protrusion (Descemetocoele) on the corneal surface. The bleb, which in diffuse light appears like a dark glistening blister, is surrounded by a circular area of infiltrated opaque tissue. Over the bleb, the distorted optic section appears as a single gray line. By retro-illumination, the bleb exhibits a yellowish glow, surrounded by a dark obstructive area.

As previously mentioned (page 115), when employing optic section in the examination of pathologic changes, it should be remembered that optical distortions of a normal posterior face can be caused by refractive and refractile effects of superficial depression or elevations (edema or blebs).

Chapter Fourteen

INFLAMMATORY LESIONS OF THE CORNEA

IN the cornea the earliest and most minute inflammatory changes can be seen with the biomicroscope until they become obscured by surrounding reactions. While differences may appear in site, structure, grouping, vascularization, and presence or absence of ulceration, the biomicroscopic appearances of the fundamental alterations of the cornea in both the noninflammatory and inflammatory lesions are indistinguishable. This is due to the fact that the avascular, laminated, transparent cornea reacts to insult according to a definite pattern. This reaction is largely governed by the biochemical and biophysic properties of the cornea and, to a lesser extent, by the type of insult. Therefore, individual alterations such as edema, opacities (infiltrations), folds or secondary vascularization, are not pathognomonic of any one disease. However, the peculiar anatomic structure of the cornea leads to a predilection of certain disease agents for certain portions of it and to the formation of lesions typical of the disease. For instance, severe conjunctival disease, such as trachoma, tends to progress in the subepithelial layers of the cornea along Bowman's membrane which acts as an entrance track as well as a barrier to deeper extension. When Bowman's membrane is destroyed (as in rosacea keratitis), the disease process may then invade the deeper layers and produce complicated pictures, in which both superficial and deep lesions are found side by side. Likewise, infiltrations which are initially deep-seated (interstitial keratitis) may migrate anteriorly. In addition, it must be realized that certain disease agents have a tissue predilection, which determines the portions of the cornea they attack; for example, herpes virus affects

nerve endings. At other times it may happen that the initial site of involvement has either been overlooked or has disappeared at an early stage in the disease. An example of this occurs in metaherpetic keratitis, which may become localized in the deeper corneal layers, the original superficial point of origin no longer being apparent.

Although there have been numerous classifications of inflammatory diseases of the cornea based on etiologic, pathologic or anatomic differences, it is advisable for the purposes of biomicroscopy, in the present state of our knowledge to adhere to a histo-anatomic classification; that is, to group these diseases according to that portion of the cornea in which their destructive influence predominates. In so doing, I have adopted a modified version of Duke-Elder's classification.⁶⁵

INFLAMMATIONS OF THE SUPERFICIAL ZONE OF THE CORNEA

The term "superficial inflammatory keratitis," in its modern sense, includes a number of primarily superficial corneal reactions of an inflammatory nature, varying more etiologically than in their clinical appearance. They are characterized chiefly by epithelial and subepithelial changes. Many of the superficial inflammations are now thought to be caused by viruses or to be associated with some manifestation of bacterial allergy. In other instances, the alterations are definitely related to systemic diseases, such as leprosy or tuberculosis (pages 509, 515), or even to the irritating action of physical agents (chemicals, dust, ultraviolet rays, and the like).*

The elusive character of these changes makes careful study with the biomicroscope obligatory. They may vary from minute epithelial droplets or vacuoles, fine punctate foci of the epithelial surface, intra-epithelial opacities, or epithelial erosions to changes in Bowman's membrane or the subadjacent stroma.

The exact localization of minute epithelial and subepithelial (Bowman's zone) changes requires careful use of the narrowest optic

* A special type of superficial punctate change has been noted by Dr. E. B. Gresser¹³⁵ occurring in four cases as part of Milian's "ninth day erythemia." All four were under trivalent arsenical therapy. Three of these cases were syphilitic and one suffered from Vincent's infection of the mouth.

section with high magnification. Many small relucet dots which appear to be on the corneal surface in the parallelepiped are found to be situated in or beneath the epithelium when examined by optic section (Fig. 209). Staining of the precorneal film line with fluorescein assists not only in exact localization but also reveals whether there is an actual connection with or a break in the surface epithelium. In herpetic keratitis, which begins superficially, reactions in the deeper layers may occur; for example, relucet parenchymal areas, folds in Descemet's layer, and endothelial precipitates. Likewise, the degree of accompanying conjunctival injection varies greatly, depending on the type and severity of the inflammatory condition. In some cases the corneal lesions are not accompanied by pericorneal injection or vascular invasion and the effects are minimal and transient. In others, recurrences with or without permanent scarring and vascularization in the involved areas may result.

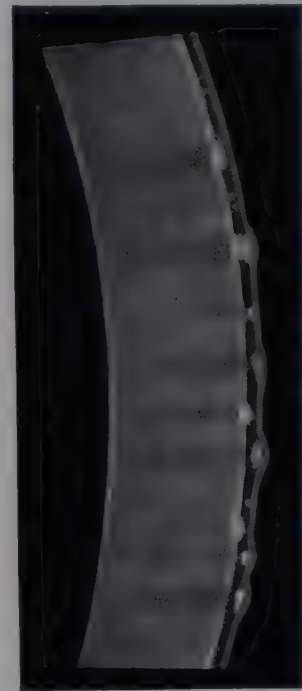


FIG. 209. Intra-epithelial deposits in superficial punctate keratitis.

There is a tendency for certain disease processes in the conjunctiva to spread over the cornea, possibly owing to the fact that the conjunctival epithelium, modified at the limbus, continues uninterruptedly over the cornea. With the milder forms, Bowman's membrane seems to act as a barrier to deeper extension; for example, in simple or catarrhal conjunctivitis, fine, more or less generalized relucet intra-epithelial dots may be seen in direct focal illumination. Likewise, in vernal catarrh the surface of the cornea may be sprinkled with fine bluish gray dots, arranged in chain or stellate aggregates. At times, such opacities may be mistaken for mucus particles, but the latter are readily displaced by eyelid movements. More extensive corneal involvement in the severe conjunctivides, such as gonococcal ophthalmia, keratoconjunctivitis eczematosa, trachoma, and acne rosacea, is, of course, well known.

In the debilitated and the aged, superficial marginal infiltration of

the cornea in an acute form with or without catarrhal conjunctivitis, may occur as discrete or punctate foci near the limbus. These infiltrations are usually found in Bowman's zone near the end of the superficial limbal spur. Softening and desquamation of the overlying epithelium results in ulceration (catarrhal marginal ulcer). In the early stages the appearance of this infiltration is similar to that of rosacea and phlyctenular keratitis, which, however, soon become vascularized and advance centrally. In rosacea, the associated skin and conjunctival involvement also assists in differentiation. The marginal corneal changes in trachoma and vernal catarrh have been treated in the chapter on the conjunctiva.

Despite the similarity of the biomicroscopic appearance of many superficial keratitides, actual variations in their clinical course do occur and in order to evaluate them properly it is necessary not only to obtain a thorough case history but also to keep each case under observation for a long period of time.

SUPERFICIAL KERATITIDES

Superficial Punctate Keratitis (Fuchs). In 1889, Fuchs described this entity as consisting of extremely minute superficial dots, which in a few days grow to form discrete, grayish rounded spots. This condition, either unilateral or bilateral, usually follows catarrhal conjunctivitis or upper respiratory infections. The punctate corneal lesions appear a week after the onset of conjunctival symptoms.

Since Fuchs' original description, a number of superficial punctate keratitides, varying slightly from one another morphologically, have been described under a variety of names. Graves states that "the term superficial punctate keratitis should be regarded merely as a noncommittal one to describe the manifestations of more than one etiologic condition."¹³² Evidently, the presence of discrete superficial opacities is pathognomonic of no special disease entity but may result from lesions of diverse etiology. Likewise, it is unknown why, in some patients, the epithelium remains unaffected; while in others, vesicles or erosions form; or in still others, the deeper parenchyma may become involved. All of these changes may represent a non-specific reaction of the anterior layers of the cornea.

While the characteristic lesion is punctate in form, in most cases coalescence of these initial minute lesions forms numerous larger spots of variegated shapes (Plate XXXI, figs. 3, 4). These, in turn, may become confluent or may remain as isolated foci, situated in the central portions of the cornea, and separated from one another by transparent intervals. The peripheral areas of the cornea generally are unaffected, although Nuel²³¹ reported a case in which the lesions were at the limbus. The corneal surface is smooth and ordinarily does not stain with fluorescein.* Only rarely have instances of epithelial roughening with tiny erosions been noted. Sclerotic scatter reveals the distribution of the lesions. By retro-illumination the larger spots (not being entirely opaque) seem to consist of minute semi-transparent droplets (Plate XXXI, fig. 5). They are localized and seen best in optic section, appearing to be composed of grayish white finely granular deposits, situated in or more often just below the epithelium (Fig. 209). Cases have been described in which the opacities were in the deeper parenchyma, associated with transient corneal edema, folds, and pigment deposits on the posterior corneal surface. Fuchs¹⁰⁵ believes that the deep form is related to disciform keratitis.

Although the disease may last for a long time with remissions and relapses, as a rule it tends to heal without recurrence. It is important to differentiate it from "pure" epithelial keratitis (page 456). An epidemic form was studied in great detail by Wright,³⁴⁰ in India; he stressed the numerous variations in its clinical appearance. In some of his severe cases, involvement of the deeper layers was so marked that keratitis disciformis was simulated; this tends to confirm the view held by Fuchs. Verhoeff likewise supports the view of the interrelationship of superficial punctate keratitis and keratitis disciformis; he believes that they are manifestations of a neuropathic disturbance, secondary to a toxic lesion of the gasserian ganglion. Trantas described a transient superficial punctate keratitis occurring in measles.

Hamilton¹⁴³ in a survey of ninety-two cases of superficial punc-

* In some cases (Plate XXXI, figs. 3, 4) I have been able to stain the superficial opacities with rose Bengal solution and with repeated instillations of fluorescein.

PLATE XXXI

FIG. 1. Columns of red dots (blood pigment) above hyphemia and large keratic precipitates. Direct focal and retro-illumination.

FIG. 2. Enlarged view of a keratic precipitate shown in Figure 1 with clear tract below it.

FIG. 3. Superficial punctate keratitis. Direct focal illumination. Lesions stained with fluorescein and Bengal rose.

FIG. 4. Superficial punctate keratitis. Optic section shows stainable lesions in epithelium.

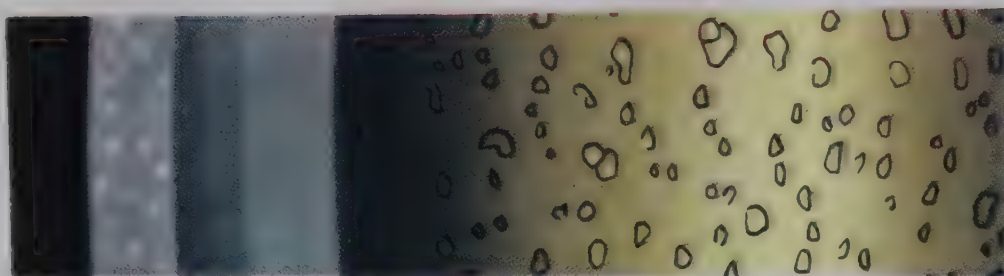
FIG. 5. Diffuse epithelial keratitis (vacuolar type).



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tate keratitis which occurred in a Tasmanian epidemic, thought this condition to be due to a virus infection of the trigeminal nerve.* This agrees with the findings of Burnet and Williams²⁵ who reported

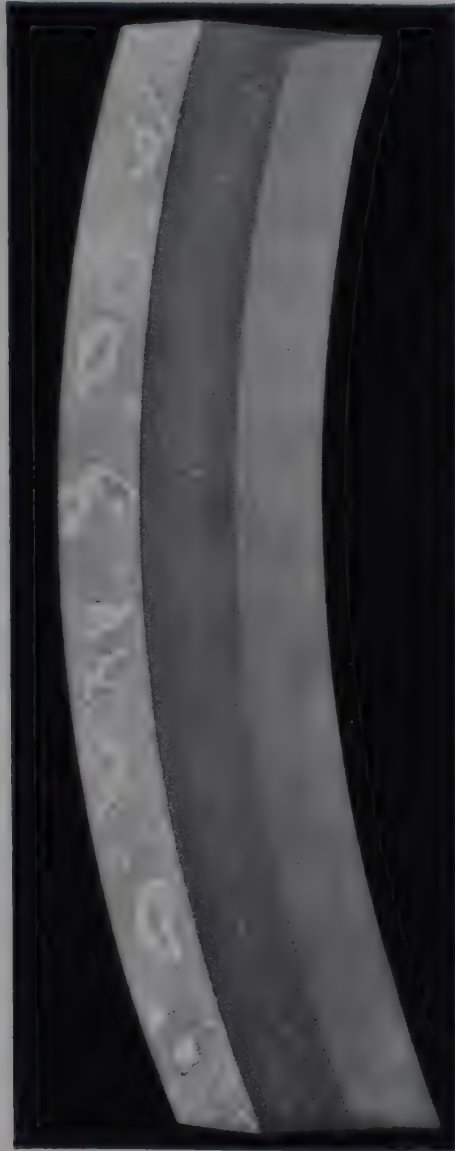


FIG. 210. Keratitis epithelialis vesiculosa disseminata in direct focal illumination.

superficial punctate keratitis in association with other herpetic lesions of the body. Hamilton found that a variety of corneal lesions attributable to one virus may occur, producing in addition to superficial punctate keratitis, multiple corneal erosions, marginal keratitis with or without ulceration, dendritic ulcer, and disciform keratitis. According to Hamilton's report, marginal keratitis was by far the most frequent of the allied conditions. He found it in seventeen cases and in four instances it preceded the superficial punctate

* Hamilton's milder cases simulate those described as acute epidemic keratoconjunctivitis (viral), page 195.

keratitis. Corneal ulceration (other than dendritic ulcers) occurred in seven cases. Although Wright, in the Indian epidemic, considered that disciform keratitis occurred as a malignant form of epidemic superficial punctate keratitis, Hamilton and Cloverdale were unable to find any cases of disciform keratitis in their series. Dendritic ulcers occurred six times in conjunction with superficial punctate keratitis. In agreement with other writers, Hamilton did not find iritis in the uncomplicated cases. The importance of repeated biomicroscopic examination with vital staining is emphasized by this investigator as a means of disclosing the presence of superficial corneal lesions because in numerous cases, although red eyes were present, gross corneal lesions could not be discovered until several days had elapsed. Superficial punctate keratitis should be differentiated from the chronic and intractable vesicular or bullous keratitis, resulting from long-standing corneal involvement secondary to chronic iridocyclitis, glaucoma, or phthisis bulbi. In all of these, vesicle formation is preceded by prolonged epithelial edema and is followed by considerable scarring.

In addition to the classic type of superficial punctate keratitis (Fuchs), several analogous varieties have been described. Among these are:

Diffuse Epithelial Keratitis (Vogt³²⁶). Vogt has reported a rare form of diffuse epithelial keratitis occurring in a young individual. The entire corneal surfaces were covered by small gray areas varying in size from 0.05 to 0.2 mm. Some were actually at the limbus. These lesions were stainable with fluorescein and were localized in the epithelium. By retro-illumination they seemed to be composed of densely grouped epithelial droplets. The surface of the cornea appeared dull. There was no vesiculation or vascularization. The patient suffered considerable photophobia and epiphora, and corneal sensitivity to touch was diminished. Slight ciliary injection was noted.

Epithelial Keratitis Associated with Iridocyclitis. Vogt,³²⁷ in 1930, described a rare type of epithelial keratitis associated with low-grade chronic iridocyclitis. The lesions consist of discrete and grayish

white opacities more or less circular in shape. These changes occur in the central portion of the cornea and raise the epithelium slightly. Each individual opacity appears to have a dense nucleus surrounded by an irregular relucet ring. Vogt³²⁶ estimated the diameter of the lesions as about 0.2 mm. The process may be transient or may last as long as a year. No erosive tendency has been reported and the lesions do not of themselves produce subjective symptoms. On the other hand, localized areas of epithelial edema are frequently associated with long-standing keratitic precipitates in chronic iridocyclitis. These areas are seen principally in the lower part of the cornea, corresponding in location to the inverted V-shaped distribution of the keratitic precipitates. After a time punctate subepithelial changes occur in Bowman's zone, which result in an increase in relucency. Vesicles and bullae may form with recurrent erosions, causing considerable pain.

*Multiple Epithelial Erosions.** Another type of superficial punctate keratitis is characterized by spontaneous and recurrent multiple epithelial erosions surrounded by narrow relucet zones. Although this condition may begin with the symptoms of acute conjunctivitis, the cornea shows a number of minute gray foci (D. F. I.), bordered by faintly hazy margins, which appear like small vesicles in retroillumination and because of an erosive tendency, stain early with fluorescein (Figs. 211, 212). After the erosions have healed, punctate spots remain which can be differentiated from those of Fuchs' type by the fact that in optic section they are connected with the epithelial surface, which may be delicately pitted. In Fuchs' type the epithelium is intact; staining rarely, if ever, occurs; the opacities lie deeper and although they are of a chronic nature there are no recurrences. In addition, it should be noted that in severe cases of Fuchs' superficial keratitis, vascularization and infiltration of the cornea may lead to dense opacification.

Multiple erosions may also appear as an occupational disease, resulting from contact with chemical vapors and dusts.

* Some of the reported cases, if it were possible to prove a dominant inheritance, may belong to the form described by Franceschetti (page 317).

Ephemeral Superficial Punctate Keratitis (Lemoine and Valois¹⁹³). Lemoine and Valois reported an acute form of superficial punctate keratitis of short duration, which they call "ephemeral keratitis."



FIG. 211. Recurrent spontaneous erosions of the cornea four days following epithelialization. A. In optic section the small irregular whitish spots in Bowman's zone seemingly are solid; their appearance by retro-illumination in B indicates their vesicular character.

This is characterized by an abrupt onset with violent pain, intense photophobia, and abundant lacrimation. The conjunctiva is markedly hyperemic. In most of the cases observed the lesions are visible only by retro-illumination; they consist of a large number of fluorescein-staining, tiny, superficial spots. Healing, with return of the normal transparency of the cornea, occurs in from twenty-four to forty-eight hours.

Keratitis Marmorata (Marble-like Keratitis). In 1930, Vogt³²⁵

reported a type of superficial keratitis, characterized by the presence of superficial discrete grayish dots, which he named keratitis marmorata (marble-like) (Fig. 213). The punctate lesions are in the

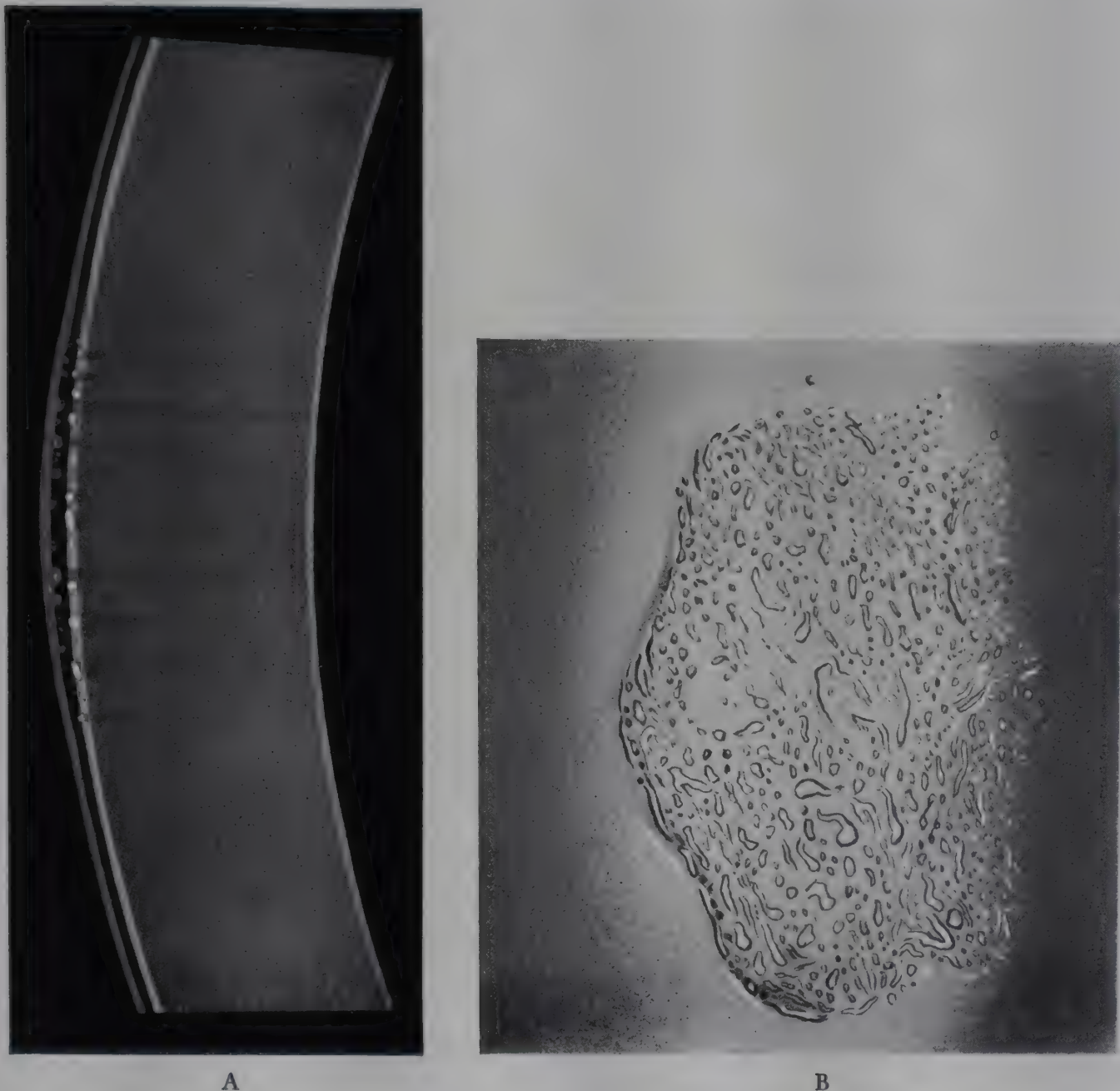


FIG. 212. Recurrent erosion two hours after an acute exacerbation. A. Optic section; note raised film line and stainable raised lesions in the epithelium and surface of Bowman's zone. B. Same lesion seen by retro-illumination indicating their vesicular character.

deeper layers of the epithelium, which at times is vacuolated. Their staining reactions vary.

Superficial Linear Keratitis. Spicer and Greeves,²⁸⁰ in 1916, described a rare form of superficial linear lesion, distinguished by an acute, painful onset and by the formation of superficial folds in Bowman's membrane (Fig. 214). This lesion may follow trauma or



A



B

FIG. 213. *Keratitis epithelialis marmorata*. A. Direct focal illumination. B. Retro-illumination.



FIG. 214. Superficial linear keratitis. (After Spicer and Greeves.)

appear spontaneously. Elevated linear ridges running in a vertical direction appear on the surface of the cornea. They have a double-tapered contour and at times assume bizarre, geometrical forms exhibiting nodelike swellings along their course. Epithelial erosion may occur in the region of these nodes, which stain with fluorescein. These lesions may heal entirely, leaving no trace of their former existence, or they may run a prolonged course, with severe permanent damage. In some cases associated hypotony may lead to atrophy and loss of the eye. It has been pointed out that these lines differ from simple folds in Bowman's membrane by the fact that the overlying epithelium does not remain smooth. In many cases this condition has probably been mistaken for a dendritic type of herpetic keratitis.

Keratitis Nummularis (Dimmer⁵⁸). This condition occurs in young agricultural workers during the summer months. Its etiology is unknown. As in the case of certain other superficial punctate keratitides, the symptoms come on suddenly, simulating acute conjunctivitis. Aust⁶ divided the course of this condition into three stages. The first stage is marked by grayish, stippled, disklike opacities in Bowman's zone, over which the epithelium is slightly raised. In the second stage the lesions flatten and have a whitish central area surrounded by a uniformly gray halo. In the third stage the halo disappears leaving a sharply demarcated, depressed facet. The lesions vary in number from one to fifteen. Occasionally they may be found deep in the parenchyma. The condition runs a chronic course without ulceration. Vascularization may occur in some instances. Although most of the reported cases were monocular, Elwyn⁸⁰ described three, occurring bilaterally, in which the lesions were situated in the anterior third of the cornea and were depressed and without vascularization. He believed that his cases belong to the third or healed stage.

Herpetic Keratitis. Herpetic infection of the cornea may manifest itself as a simple type (herpes simplex corneae) or as the ocular moiety of a herpes zoster.

Herpes febrilis keratitis (simplex). Herpes simplex corneae may

occur as an isolated corneal lesion or in association with herpetic lesions of the lips, genitalia, and so forth. Corneal involvement may also occur during a hyperthermic systemic disease (e.g., upper respiratory tract infection, pneumonia, or malaria) or with artificially induced hyperpyrexia (foreign protein shock therapy and short-wave diathermy). I reported a case of herpes simplex corneae following induced hyperpyrexia employed in the treatment of rheumatoid arthritis.²⁴

The lesion appears as a superficial infection in the epithelium (involvement of the nerve-endings); it is composed of groups of fine epithelial dots or small vesicles. The development of fissures and erosions leads to the formation of the so-called dendritic ulcer, which is the most characteristic manifestation of simple herpetic infection of the cornea.

Herpes simplex corneae may manifest itself in several ways, depending on the virulence of the virus, or the resistance of the tissue. On biomicroscopic examination the initial process may appear as a localized faint nebulous haze in the epithelium or as groups of punctate whitish intra-epithelial dots. In some instances its presence may first be recognized by a small area of localized epithelial bedewing.* In others, however, the appearance of the fine epithelial opacities may be followed by development of small clear vesicles in the epithelium, with the underlying Bowman's zone slightly translucent. The vesicles may heal leaving only a minimal haze in Bowman's zone or no residuum. New vesicles may appear before the first crop has completed its cycle. The corneal nerve fibrils are markedly visible when viewed in direct focal illumination. At times,

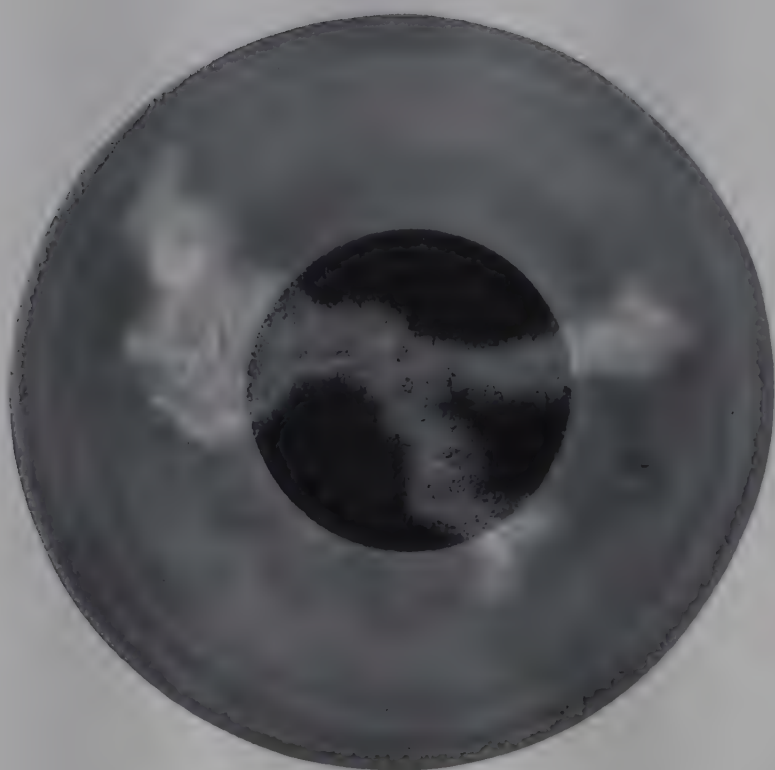
* Wetzel³³⁷ recently stated: "Ryan doubts the existence of original vesicles. He remarks that it seems odd that the vesicles should all occur and erupt at the same time, that they do not present themselves in crops or occasionally one at a time when we could observe them while we are watching the corneal lesion. Gundersen, who made a special study of 227 cases of herpes corneae and says positively, 'Not a single case of vesicular herpetic keratitis, described in text-books as an acute eruption of very fine vesicles distributed over the cornea, has been seen by me in the course of this study,' is of like mind.

"However, the diagnosis can be made readily, even if no vesicles are to be found. The patient usually comes complaining of having gotten something in his eye which his own efforts and those of his family have failed to locate. The examiner will also fail to find it, and it is just here that the diagnosis may fail, too, for unless the cornea be stained and examined with the slitlamp, the tiny erosions may entirely escape notice."

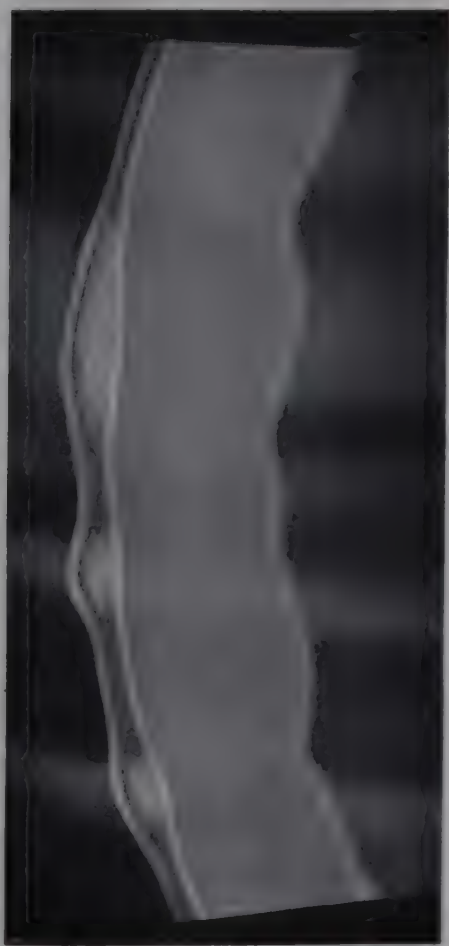
the vesicles may coalesce, rupture, and form an irregular ulcer. The smallest lesions stain with fluorescein even though gross solution of continuity of the epithelium is not perceived. Actual solution of continuity usually results in the formation of fissures or cracks. In optic section, the epithelium on each side of a delicate fissure or crack (appearing as a break in the precorneal film line) (Fig. 164) is swollen and even raised. The borders appear hazy because of underlying relucency. The subjacent Bowman's zone may be thickened and relucant. When these fissures fuse and spread branch-wise, a characteristic dendritic or arborescent figure is formed. This lesion constitutes the so-called dendritic ulcer (Fig. 215; Plate XXXII, fig. 1).

Each branch of the dendritic figure is composed of an irregular fissure, the borders of which consist of swollen, raised and hazy epithelium. The ends of the branches are frequently club-shaped. In the neighboring areas, but unconnected with the main lesion, circularly grouped punctate aggregations may be seen in Bowman's zone. When fully formed, these aggregations stain beautifully. It is possible to stain the central, more necrotic areas with methylene blue or azur ii and the surrounding affected epithelium with fluorescein. Owing to the elevation of the surrounding epithelium (epithiolysis), the fluorescein infiltrates beneath the epithelium for a considerable area around the ulcer. In most instances the ulcers tend to remain localized until healing processes begin (Fig. 216), but at the same time, deeper reactive changes are noted. There is usually definite thickening of the underlying cornea, as demonstrated by posterior bulging of the optic section. Small areas of relucency may be seen in the deep stroma, accompanied by tiny linear opacities and dark spaces; or a circumscribed disciform keratitis may develop. Folds in Descemet's membrane with or without keratic precipitates, directly behind the figure itself, indicate the degree of severity of the attack. However, it should be noted that even in mild cases, fine precipitates frequently appear on the posterior corneal surface behind the lesion.

While the disease may be prolonged and relapses are common,



A



B

FIG. 215. Herpetic lesion of the cornea; later stage. A. Diffuse illumination. B. Optic section.



FIG. 216. Superficial herpetic scar.

ultimate healing is the rule. Mild vesicular lesion, involving only the epithelium, may heal without scar formation. However, when Bowman's membrane is involved, which usually occurs in the dendritic

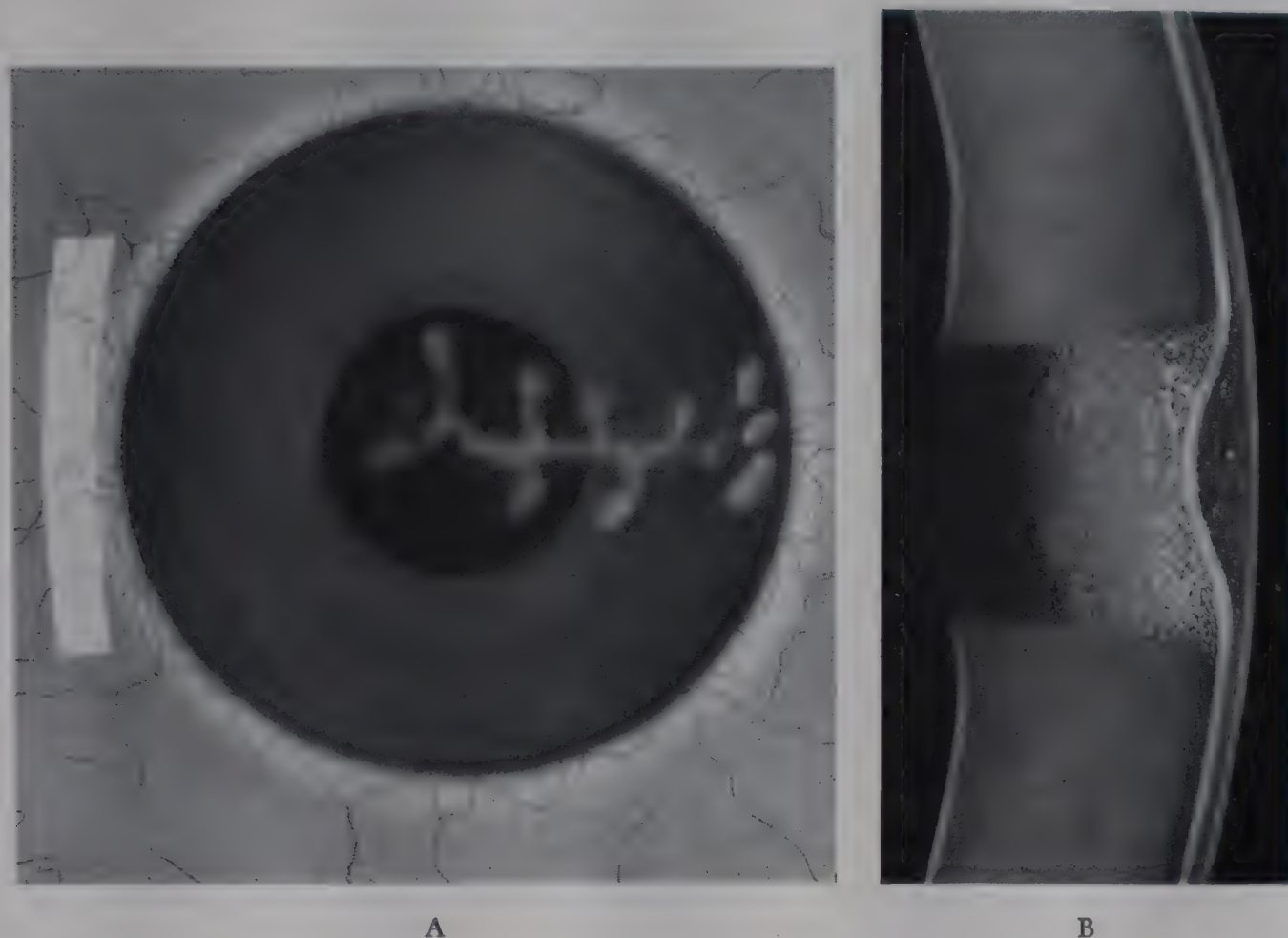


FIG. 217. A. Herpetic scar of the cornea by sclerotic scatter. B. Optic section through a portion of the lesion.

or ulcerative types, healing results in characteristic dendritic or grouped roundish scars in Bowman's zone (Fig. 243).

Coalescence and extension of the ulcerative process or secondary infection is signified by a change from the typical herpetic appearance to a fairly large grayish yellow irregular ulcer, the edges of which are infiltrated. When the process is intense, hypopyon may occur. Healing of such a lesion is followed by a dense scar. In many instances, distortion of Bowman's membrane and thinning of the corneal thickness results (Fig. 217).

In severe cases an accompanying herpetic iritis may occur. Recurrent attacks of iritis without corneal involvement have been described.

Herpes zoster keratitis (epidemicus). Herpes zoster of the ocular tissues may result from infection by the specific neurotropic virus, or as a symptomatic nonspecific form caused by affections of the gasserian ganglion, such as syphilis, tuberculosis, meningitis, trauma, or neoplasms. The appearance of the ocular manifestations and symptoms are similar in all types and one attack tends to confer immunity.

Actual herpetic lesions may occur in the eyelid, the bulbar conjunctiva and episclera, and less frequently on the palpebral conjunctiva and cornea. During the eruptive stage, involvement of the eyelids may cause considerable swelling and tenderness. This may be attended by edema of the conjunctiva and corneal epithelium. The corneal lesion is characterized by a granular subepithelial infiltration in Bowman's zone or in the anterior parenchyma. These lesions are disklike, either single or multiple.

Usually deeper changes are perceived in the parenchyma, as irregular opacities, edematous clefts, and folds in Descemet's membrane. These alterations cause an increase in parenchymal thickness, commonly seen as a posterior bulging. In some cases, the overlying edematous epithelium forms vesicles, which break down and lead to stainable erosions and ulcers. In others, no ulcerations occur and all acute symptoms may abate within a month's time. Depending on the intensity of involvement, some degree of permanent scarring and opacification always occurs, resulting in variations of corneal thickness. In severe ulcerative cases, vascularization of the cornea may follow (Fig. 218).

Occasionally, a nodule may develop in the episclera and sclera (scleritis). This is accompanied by severe conjunctival injection and edema. It appears as a raised gelatinous node, dark at the center. In optic section, staining with azur ii and fluorescein shows a dark bluish necrotic center with a dimpled green film line. Healing may be followed by a permanent, slightly depressed, dark slate-colored scar beneath the conjunctiva (Fig. 134).

As a rule, the acute phase is accompanied by some degree of iritic irritation. This is manifested by an aqueous flare and by keratic

precipitates. As in herpes simplex the deposits may be few, and restricted to an area directly behind the corneal lesion; but if plastic iritis supervenes, a heavy exudation forms. Severe herpetic iritis

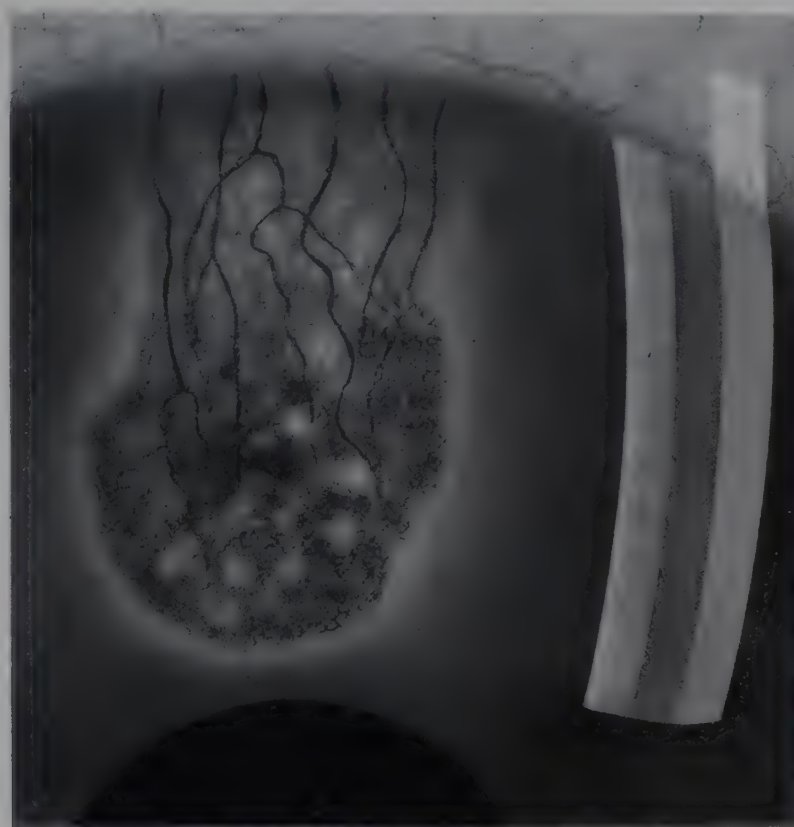


FIG. 218. Corneal scar (vascularized) in herpes zoster by retro-illumination.

may result in characteristic punched-out atrophic patches in the iris. A lesion of the gasserian ganglion in herpes zoster with consequent destruction of the nerve pathways (indicated by anesthesia of the cornea) may lead to the development of neuroparalytic keratitis.

Keratitis metaherpetica. A transition form of herpes cornea, known as keratitis metaherpetica, follows healing of a dendritic ulcer. The arborescent figure of dendritic ulceration is not seen, but small, round or oval ulcers with irregular borders are noted. These are usually single, and the bordering epithelium is not raised, in contrast to the dendritic type of typical herpes simplex. Vogt³²⁷ described a deep parenchymatous keratitis, closely resembling keratitis disciformis, which ensued several weeks after an attack of herpes simplex. In this type the epithelium is unaffected except for edema directly over the lesion. The opacity occurs in the anterior stroma,

which is swollen and may be accompanied by folds in Descemet's membrane. Similarly, Schnyder²⁷⁰ described, in association with recurrent labial herpes, a herpetic lesion of the posterior corneal face, characterized by conspicuous vesicle formation in the midst of an opacity on the posterior surface, which interfered with the specularity of the endothelial layer.

Vaccinial Keratitis. Vaccinial keratitis may result from accidental inoculation of the cornea. This has occurred in children following vaccination. It may manifest itself either as a slight subepithelial infiltration, as a superficial vesiculation and pustulation, or as a disciform keratitis. Perera²³⁴ described a case which began with a lesion on the upper eyelid; two weeks later this was followed by a superficial corneal ulcer in the pupillary area (Fig. 219). This lesion was succeeded by a disciform keratitis, in which a grayish white ring formed in the deeper layers. There were folds in Descemet's membrane and deposits on the posterior corneal surface. A faint corneal scar remained.

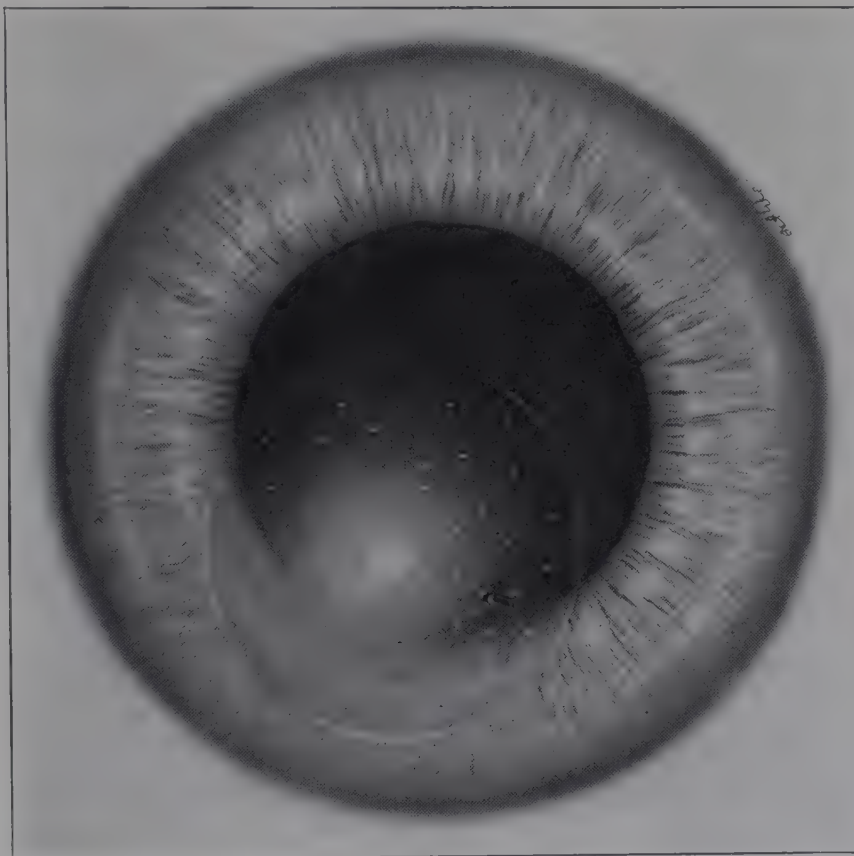
Varicella. There are only a few reports of inoculation of either the conjunctiva or cornea by the virus of chickenpox. Isolated vesicular formation with or without marked ulceration has been noted. Little scarring occurs. In one case a circumscribed parenchymatous lesion with posterior bulging followed a vesicle. Healing was attended with practically no scarring.²³⁹

KERATITIDES ASSOCIATED WITH PHYSICAL INFLUENCES, METABOLIC AND NEUROTROPHIC DISORDERS

Interference with normal nutrition and health, owing to mechanical drying, exposure, lack of tears, or to metabolic or neurotrophic disorders, results in a number of special corneal affections, primarily superficial. Secondary infections superimposed on the epithelium, devitalized from any of the aforementioned causes, intensify the severity of these conditions and result in varying degrees of corneal destruction. Rosacea and phlyctenular keratitis are included under this heading, although the former is associated with skin lesions and the latter with a state of sensitization to some



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B

FIG. 219. Vaccinial keratitis. A. Lid lesion. B. Corneal lesion. (From Perera.)

endogenous toxin. However, the underlying basis for both these conditions may be some metabolic defect.

Keratoconjunctivitis Sicca. This disease, generally bilateral and of long duration, is characterized by failure of the normal lacrimal secretion, possibly owing to some affection of the lacrimal gland. It results in drying of the conjunctival and corneal epithelium with serious sequelae. It must be differentiated from xerosis, which may develop even in the presence of normal lacrimal secretions. Sjögren believes that the ocular symptoms are probably part of a larger symptom-complex, occurring chiefly in women after the climacteric, and associated with xerostomia, rhinitis sicca, pharyngitis sicca, laryngitis sicca, and chronic polyarthrititis. However, only part of the symptom-complex may be present in any one patient. He considers the associated clinical features to be typical of chronic infection or toxemia but the possibility of endocrine disorders and avitaminosis must also be kept in mind.

With the biomicroscope the precorneal film in the lower part of the cornea is seen to be altered from its normal free-flowing watery state to a viscid mucoid film, containing flakes of epithelium and mucus (Plate XXXII, figs. 2, 3). The precorneal prow-line of tear fluid at the lower eyelid margin reveals numerous fine mucoid particles and epithelial debris. A few filaments composed of mucoid material may be adherent to the corneal surface. The occasional presence of these filament-like mucoid threads and flakes has caused it to be mistaken for filamentary keratitis. In true filamentary keratitis (*Fädchen* keratitis) (Plate XXXIV, fig. 5) the filaments are fewer in number (8 to 10), long, twisted, and more firmly attached to the corneal surface; while in keratoconjunctivitis sicca the filament-like mucoid threads and flakes are smaller and more numerous, have a frothy appearance, and are movable (Plate XXXII, fig. 4).

Since filamentary proliferations may occur after herpetic lesions, recurrent erosions or abrasions, their presence cannot be considered as pathognomonic of this condition. Bruce³⁹ reported fourteen cases of keratoconjunctivitis sicca, only two of which revealed the presence of true epithelial filaments.

Sjögren²⁷⁵ stated that "in the outer layer of the epithelium small roundish gray foci are visible, together with small epithelial flakes resembling fluted tissue paper, and between there is a tiny, pin-point stippling. The most common feature is the gray epithelial foci, which are never entirely absent." Retro-illumination readily discloses the varied forms of the filament-like mucoid deposits and tiny epithelial stippling (Fig. 220; Plate XXXII, fig. 4). Optic section may reveal small subepithelial relucant foci. For differential diagnosis, Sjögren recommends vital staining with a 1 per cent aqueous solution of rose Bengal (Plate XXXIII, figs. 1, 2). The bulbar conjunctiva and the corneal flakes are thus stained a deep red in the exposed interpalpebral zone. A pathognomonic bright red triangular area is found on either side of the partially reddened cornea. With high power (40 \times) the stained areas consist of cells faintly outlined but with deeply colored nuclei. Corneal vascularization and even pannus formation of the lower cornea have been noted. The palpebral conjunctiva, likewise, may be injected to a varying degree, especially in its lower portions. Sometimes slight edema of the bulbar conjunctiva may be present with dulling of its normal luster, but advanced dryness (prexerosis) is infrequent. Rarely, changes in the deeper parenchyma may occur in the form of small grayish relucant areas. Despite the extent of the epithelial changes, corneal ulcers are uncommon.

Keratitis e Lagophthalmos. Exposure keratitis is associated with orbicularis oculi paralyses, induced by affections of the facial nerve, or with faulty closure of the eyelid in exophthalmos, ectropion, eyelid cicatrices, and coma. The imperfect closure of the eyelids leaves the mucous surfaces in prolonged contact with the air and results in overevaporation of the tear fluid with consequent desiccation of the epithelium. Degeneration of the epithelium is followed by xerosis. The early biomicroscopic picture usually seen is that of a delicate punctate epithelial haziness, appearing especially in the lower parts of the cornea as a horizontal band of epithelial edema and vesicles with eventual fissuring and exfoliation of the epithelium (Plate XXXIII, figs, 3, 4, 5). Fluorescein staining of the

PLATE XXXII

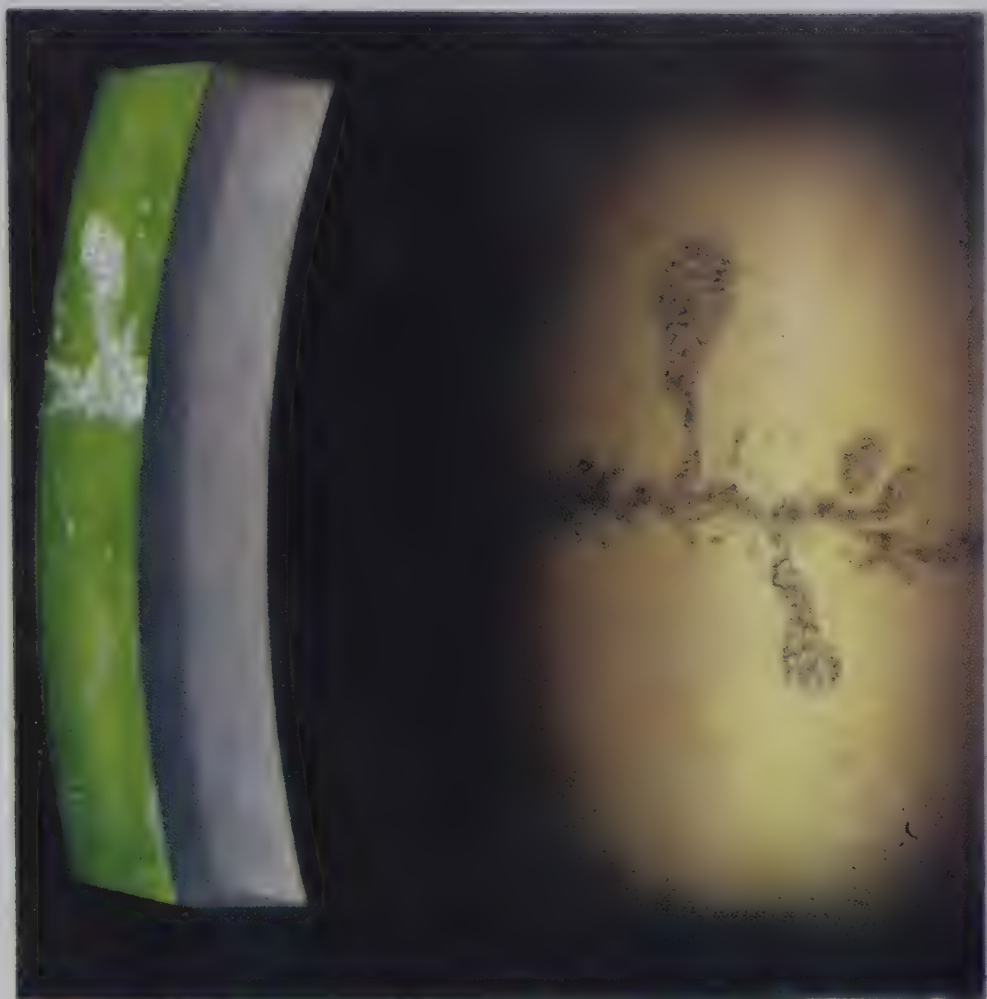
FIG. 1. Herpes corneae (Early). Direct focal illumination and retro-illumination. Portion of lesion which is shown in parallelepiped was not stainable.

FIG. 2. Keratoconjunctivitis sicca, fluorescein staining of changes. Adherent mucoid material is seen on the surface of the corneal parallelepiped.

FIG. 3. Keratoconjunctivitis. Appearance of changes in optic section. 40 \times .

FIG. 4. Keratoconjunctivitis sicca. Filament-like mucoid deposits on the corneal surface by retro-illumination.

FIG. 5. Keratoconjunctivitis sicca, stained mucoid flakes on the anterior corneal surface in direct focal illumination.



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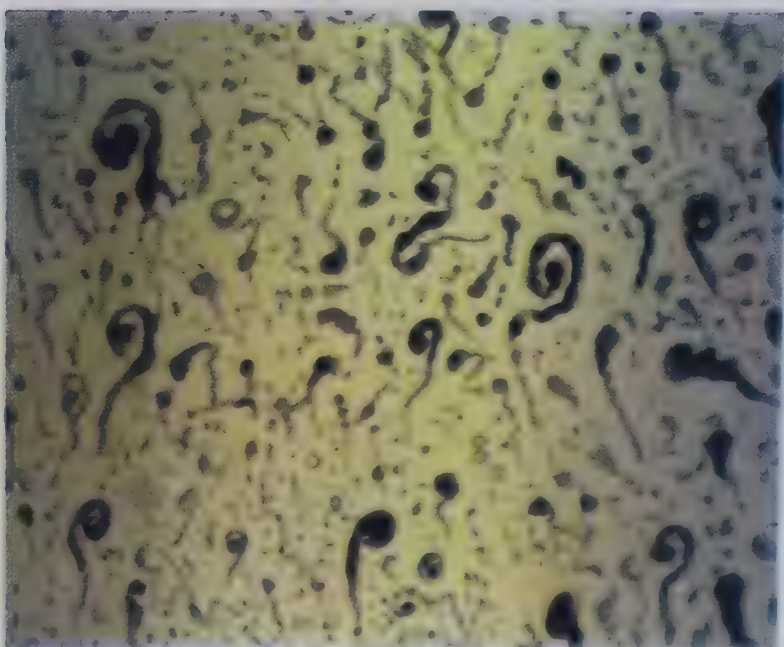




FIG. 220. Various forms of filamentary mucous threads.

lesions occurs early in the development of this condition. The resulting corneal opacity is usually found at the point where the lower eyelid comes in contact with the cornea. In some respects the scar may resemble that of bandformed keratitis.

In severe cases, unless remedial measures to protect the cornea, such as bandaging, tarsorrhaphy, or plastic surgery for correction of ectropion, are instituted, the epithelium becomes dry, opaque and flaky (xerotic) and finally keratinized.

At the same time, the anterior parenchyma may become relucet and infiltrated. Secondary infection with ulceration may eventuate in hypopyon, perforation of the cornea and panophthalmitis.

Acne Rosacea Keratitis. This disease of unknown etiology,* characterized by remissions and relapses, is nearly always associated with acne rosacea of the face, and most often follows involvement of the eyelids or conjunctiva. However, the cornea may become involved without the concomitant presence of facial lesions. The disease is usually bilateral, although it may appear in one eye before the other. It is more prevalent in women than in men.

Acne rosacea keratitis may manifest itself as a mild marginal involvement, indistinguishable from the so-called acute marginal lesions which are seen in catarrhal or phlyctenular keratoconjunctivitis (page 481). These marginal subepithelial infiltrations may heal without ulceration or marked vascularization, but they usually leave faint nebulae adjacent to the limbus.

The more severe forms of rosacea of the cornea are generally associated with facial lesions of varying degrees of severity. Although the character of the infiltration may vary from case to case, in most instances two principal forms are observed. One is the sclerocorneal type and the other is the nodular type. The former may start as a tenuous subepithelial infiltration, localized to one or more portions of the limbus. This infiltration is accompanied by vessels derived from the perilimbal vascular arcades and tends to progress toward the center of the cornea. At first, the lesions appear

* Johnson and Eckardt's work¹⁶¹ has suggested that there is possibly some relation between riboflavin deficiency and rosacea keratitis.

rounded or ovoid but become more irregular as they approach the pupillary area. Later, they coalesce to form a triangular highly vascularized opacity, the apex of which approaches the pupillary area. With the narrow beam, the infiltration in these areas is seen to penetrate the parenchyma to varying depths. Vascularization is proportional to the intensity of the corneal involvement. The softening of the corneal tissue permits deeper invasions of the accompanying vessels. The subepithelial infiltration migrates into the deeper layers of the parenchyma, accompanied by the now enlarged and freely anastomosing vessels. At this stage, the optic section shows an increase in corneal thickness and retro-illumination reveals epithelial edema. The latter may progress to the formation of vesicles which, on rupturing, result in stainable erosions and ulcerations. Such ulceration may occur in the limbal regions or over the most dense portion of the infiltration. There is a tendency for the ulcerations to recur so that concomitantly healing areas and fresh lesions may be seen side by side. Eventually, after repeated attacks the whole cornea may become irregularly scarred and vascularized, its surface uneven and faceted. In the quiescent stages the epithelium regenerates and covers the scarred and irregular underlying zones.

In the nodular type, circumscribed infiltrations occur in the form of dense conglomerates. One or more isolated nodules may develop in any portion of the cornea but the common site of predilection is usually near the limbus (Plate XXXIV, fig. 4). In the beginning, they are subepithelial in and below Bowman's zone but later may become several millimeters in size and extend deeply in the parenchyma. As the condition advances, superficial staining erosions and ulcers (chalky-white bases) develop, causing surface irregularities; at first they are grayish in color but later they tend to become whiter and more relucet having denser, opaque centers. The presence and sites of such lesions may be demonstrated early by sclerotic scatter (Fig. 221).

A fascicular type of vascularization generally accompanies these nodular infiltrations. The vessels may be large, several millimeters in length, and terminate by arborization within and around the

PLATE XXXIII

FIG. 1. Keratoconjunctivitis sicca. Typical staining with rose bengal. A pinkish triangular patch occurs on either side of the cornea, also small pinkish staining flakes on the corneal surface.

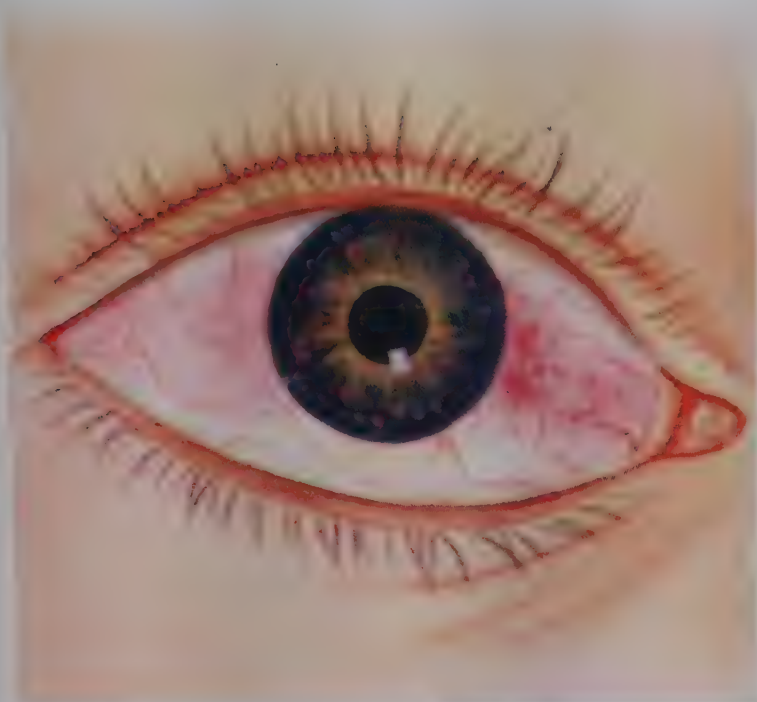
FIG. 2. Keratoconjunctivitis sicca. Optic section through the conjunctiva in the areas stained with rose bengal. Adherent mucoid particles and desquamated epithelial cells take deeper pink stain. Film line stained afterward with fluorescein.

FIG. 3. Keratitis e lagophthalmos. The location of the corneal lesion.

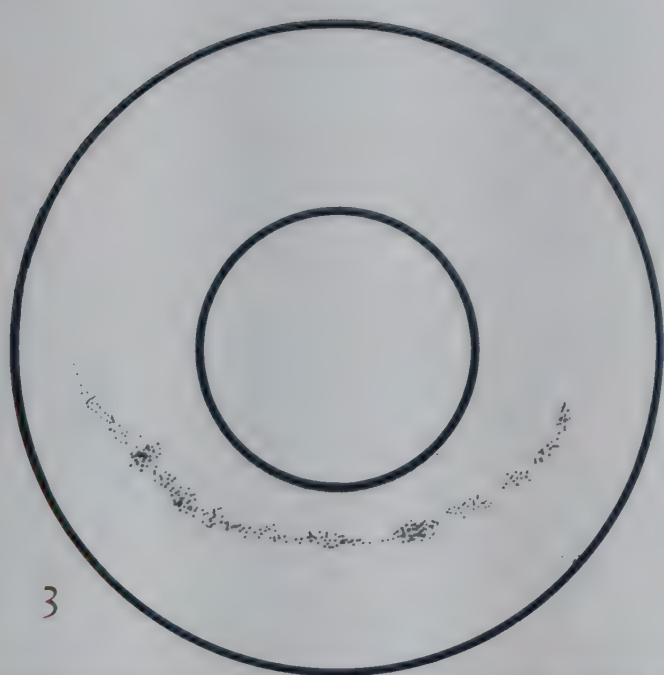
FIG. 4. The above opacity (Fig. 3) in direct focal illumination stained with fluorescein.

FIG. 5. Same lesion (Figs. 3 and 4) in optic section.

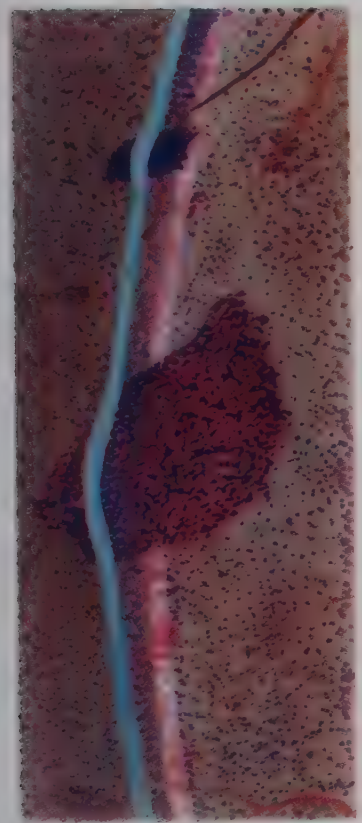
FIG. 6. Marginal phlyctenular ulcer. Diffuse illumination.



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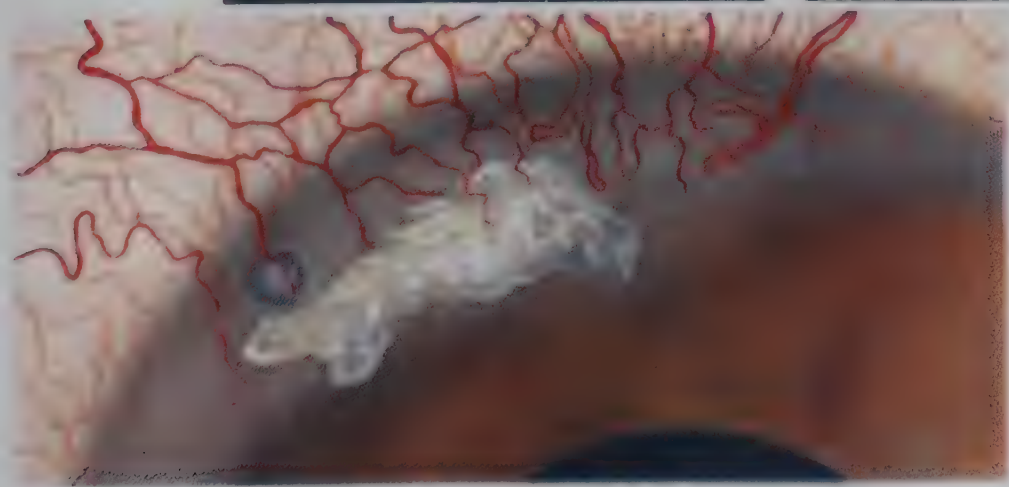
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6

lesion. This picture resembles that produced by severe phlyctenular keratoconjunctivitis. Depending on the severity and extent of the disease, permanent scarring and vascularization results. With each



FIG. 221. The typical lesions of acne rosacea keratitis in sclerotic scatter.

succeeding attack, new nodules may form or ulcerations of apparently healed pre-existing ones may occur, so that eventually the entire cornea becomes scarred and vascularized. The areas between the lesions may be occupied by small subepithelial punctate infiltrations. When examined in direct focal illumination in the quiescent stage, considerable distortion and thinning of the cornea is observed in the involved areas. The scars may be superficial or they may involve all the layers of the parenchyma and by diffuse illumination or sclerotic scatter may be seen to assume characteristic shapes, which have been described as horseshoe, tongues, or triangles. A more malignant form has been observed, in which the process is so marked at the periphery that it may be confused with *ulcus rodens*.

Phlyctenular Keratoconjunctivitis. Although this disease (keratoconjunctivitis eczematosa) may occur as a primary corneal lesion,

it is usually associated with phlyctenular conjunctivitis. In mild or ordinary forms, the lesions are characteristic but in severe forms they may be indistinguishable from keratitis associated with acute rosacea. However, it should be borne in mind that the latter occurs in adults, who usually have facial acne rosacea, while the former occurs predominantly in scrofulous children (phlyctenular conjunctivitis, page 190).

Phlyctenular keratitis is characterized by the development of a "phlycten." These phlyctens may occur at the limbus in association with conjunctival lesions or they may appear independently on the cornea. By diffuse illumination a phlycten appears as a tiny yellowish white conical elevation. By optic section the early focus is seen to be covered by an intact film line and epithelium. Shortly, ulceration occurs at the summit of the elevation, which becomes umbilicated as a result of slough. When stained with azur ii and fluorescein, the central necrotic umbilicated area appears blue beneath the indented green-stained film line. A surrounding relucet reactive zone forms a halo about the phlycten at the height of its activity.

Ulceration is usually accompanied by congestion of the neighboring limbal arcades and by invasion of one or more branches, arborizing around the lesion (Plate XXXIII, fig. 6). In this mild form healing may occur, leaving only a small faint nebula in Bowman's zone. In more severe cases, the lesion tends to migrate toward the center of the cornea. The peripheral portion of the phlycten heals, while an advancing and infiltrating apex continues to migrate centrally (Plate XXXIV, figs. 1, 2). This advancing portion is usually circular and surrounded by a faint granular haze. Small, staining erosions and ulcerations accompany this process. Eventually, its original connection to the periphery may be indicated only by a faint relucet track in Bowman's zone, extending to the limbus. Vascularization of this track occurs early. Elongated capillary loops or larger vessels extend from the limbus to the phlycten itself and arborize around it.

These vessels run in a straight course, forming a leash (fascicular keratitis). Retro-illumination may reveal neighboring epithelial

edema. After healing occurs the resultant scar is seen in the form of a band-shaped superficial opacity, which is most dense in the region of the advancing edge of the phlycten, with a fainter relucant track toward the limbus (sometimes resembling a comet).

Another manifestation of phlyctenular keratitis is pannus formation. In this case the vascularization is more widespread; in some instances it completely encircles the cornea. Multiple phlyctens usually develop toward the central portions of the cornea. The surface areas in and around the pannus are edematous and relucant, resulting in loss of luster and irregularity. Since pannus formation consists of an invasion of permanent tissue in Bowman's zone, destruction of this layer with permanent opacification and vascularization results. In advance of the pannus, denser and more relucant scars mark the sites of the healed phlyctens.

In more malignant forms, the infiltration and vascularization extend into the deeper layers of the cornea in an irregular manner and the picture may resemble rosacea keratitis or tuberculous keratitis (Plate XXXIV, fig. 3).

Further ulceration and secondary infection results in dense scarring and thinning of the cornea. The scarring, which may occur throughout the layers of the cornea, is associated with superficial and deep vascularization. In optic section, these scars may produce deep irregularities (folds) in Bowman's zone. Relapses cause extension of this scarring process until large areas of the cornea are affected and much visual damage results.

Xerosis and Keratomalacia. Xerosis corneae is a condition belonging to a larger symptom-complex found in association with malnutrition, particularly avitaminosis A; it may also occur as a secondary degeneration in severe external diseases, such as trachoma, pemphigus, or keratitis e lagophthalmos in which widespread destruction of conjunctival glands occurs. Corneal involvement ensues either as extension or as part of conjunctival xerosis (page 166).

Pillat²⁴¹ described the earliest changes under the designation of prexerosis. This stage is marked by loss of corneal luster, rapid desiccation of the corneal surface on exposure, and corneal anesthe-

PLATE XXXIV

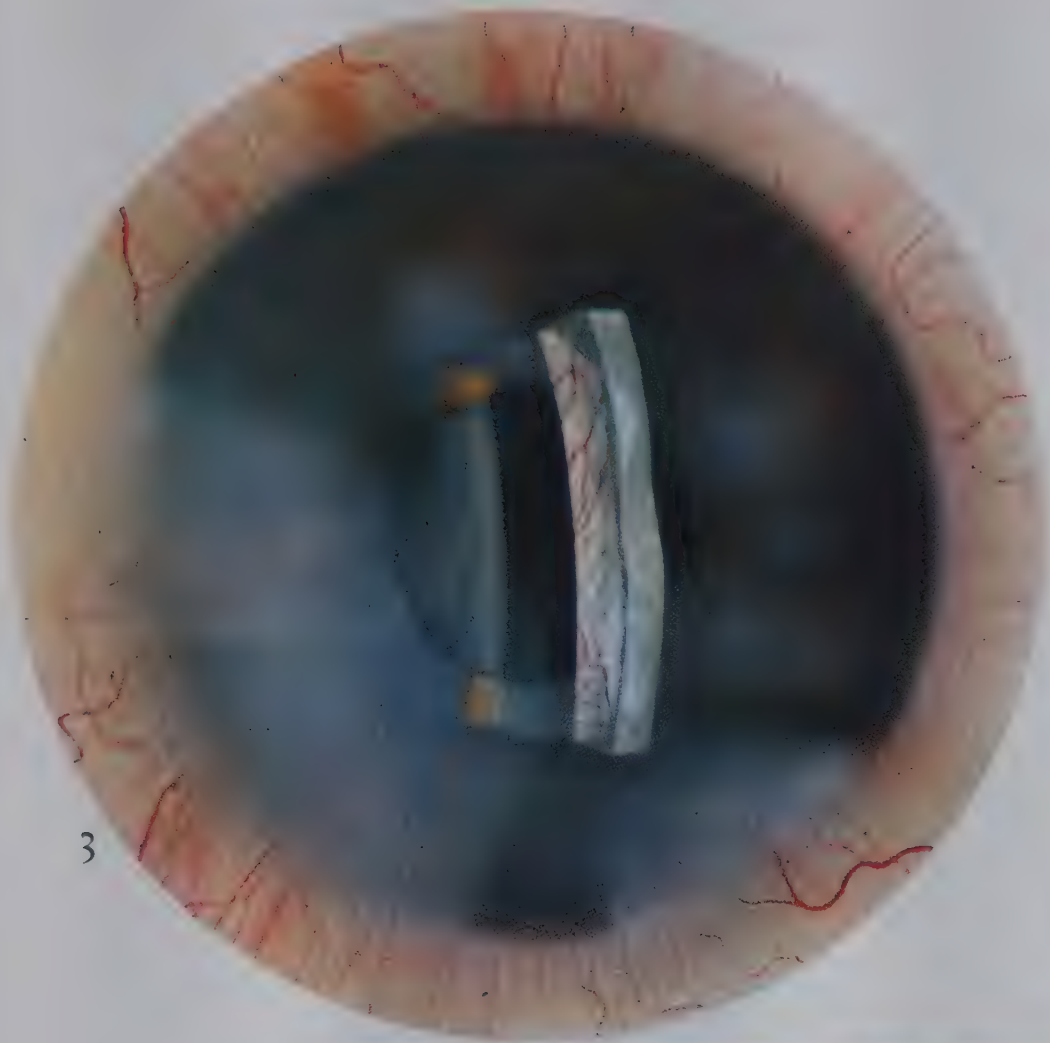
FIG. 1. Phlyctenular keratoconjunctivitis by sclerotic scatter.

FIG. 2. The same lesion (Fig. 1) by direct focal illumination showing the infiltration, surface irregularity and location of vessels.

FIG. 3. Severe form of keratitis eczematosa, illustrating superficial and deep vascularization.

FIG. 4. Acne rosacea keratitis; nodular type of infiltration.

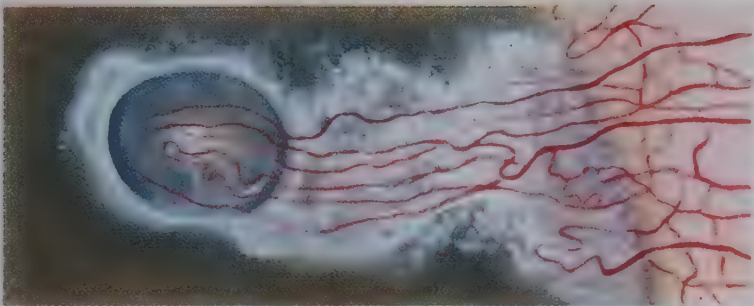
FIG. 5. Filamentary keratitis; epithelial filaments stained with fluorescein.



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sia. At this point, there is considerable resemblance to keratitis neuroparalytica. As progression occurs toward true xerosis, the lesions appear as irregular patchy confluent areas of whitish gray

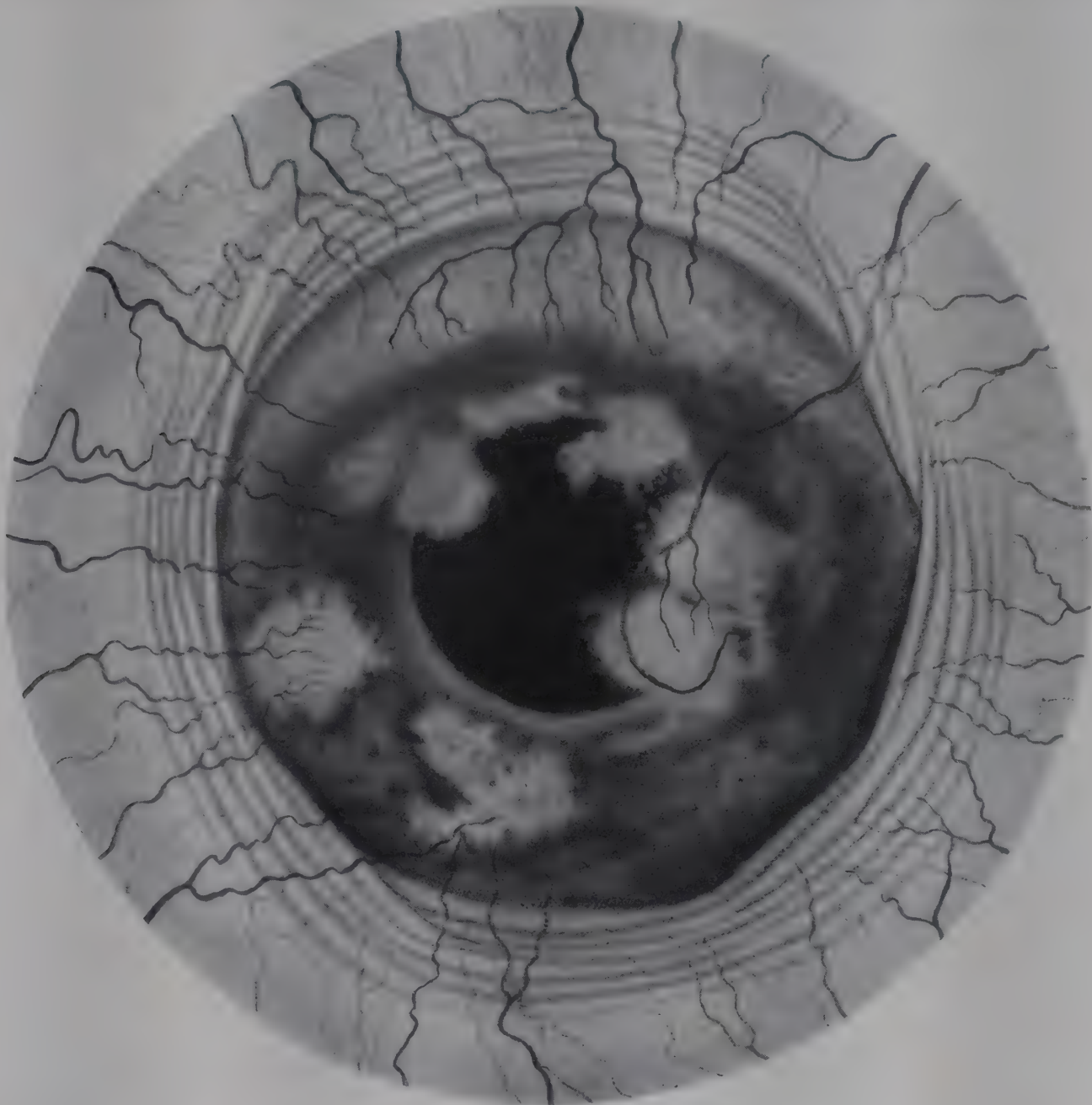


FIG. 222. Xerosis conjunctivae and corneae. Diffuse illumination.

color (Fig. 222). When these areas are on the surface, they may have a foamy appearance similar to Bitot's spots in the conjunctiva (Plate V, fig. 4). They may be continuous with neighboring conjunctival patches in the exposed interpalpebral zone or they may occur concentrically along the limbus in crescentic fashion.

The opacities are subepithelial in site. Pigmentation may take place, but is never as marked (according to Pillat) as the conjunctival pigmentation observed in Chinese suffering from avitaminosis A.

As the condition advances, the cornea becomes dull, markedly infiltrated and softened, and eventually keratomalacia develops. In this phase, the whole cornea becomes opaque, the conjunctiva becomes dry and wrinkled concentrically, and both superficial and deep vascularization occurs. Epithelial exfoliation, ulceration with hypopyon, and abscess formation are common sequelae which may result in loss of the eye following perforation.

On the other hand, in chronic avitaminosis A, continued maintenance of the prexerotic stage may lead to metaplasia of the corneal epithelium, characterized by keratinization and cuticular epithelization of the cornea similar to that which occurs following long-standing exposure due to ectropion (senile or cicatricial, following burns and the like) or lagophthalmos.

Diffuse Vesicular Keratitis. Neurotrophic disorders often lead to the formation of epithelial vesicles or bullae. Interference with the normal antidromal sensory nerve influences results in an accumulation of cellular metabolites, with consequent edema and vesiculation. Duke-Elder states that "in all cases wherein epithelial vesicles occur in the absence of acute inflammation or high tension, the neuropathic factor should be investigated."

Instances of idiopathic vesiculation of the cornea are known in which the entire corneal surface is sprinkled with minute vesicles. Large clear bullae may form in consequence of coalescence of neighboring vesicles. Rupture of these bullae leads to abatement of the affection. The condition is painful and recurrence is common. Healing may occur without leaving opacification, but in the long-standing recurrent types some degree of superficial opacification results, owing to changes in Bowman's membrane.

Neuroparalytic Keratitis. This affection follows disturbance or destruction of the trigeminal nerve supply to the cornea. The site of the nerve lesion may be ganglionic, infraganglionic, or supraganglionic. Clinically, it is often seen after operations on the gasserian ganglion or after injections for the relief of trigeminal neuralgia.

As a result of the trophic interference with the normal epithelial

cellular metabolism, certain alterations occur in these cells, especially when the cornea is unprotected. Edema (bedewing and vesicles) and relucant punctate infiltrations are the most important of these. The latter are probably due to the accumulation of cellular metabolites, such as intracellular inclusions, cellular coagula, or extracellular deposits. Concomitant vasodilation accounts for the occasional conjunctival and iridic congestion. Because of anesthesia, subjective symptoms (except for blurring of vision) are minimal, but loss of the eyelid reflex leaves the cornea defenseless against foreign bodies and mild traumas.

The clinical appearance of neuromparalytic keratitis depends on the acuteness of onset and to what extent the cornea is protected. Therefore, the changes in this disease may vary from slight epithelial alterations to severe exfoliative destruction of the epithelium, ulceration, and even necrosis of the entire cornea. In the early stages, characteristic irregular punctate relucant epithelial foci, accompanied by varying degrees of conjunctival injection, may be observed in direct focal illumination. By retro-illumination, edema and vacuolation of the epithelium are seen. If at this time the eyelids are closed to protect the cornea (bandaging or tarsorrhaphy) the condition may be arrested and the epithelium may return to its clear state. Otherwise, the process continues with the development of erosions and epithelial exfoliations of varying sizes. The appearance of the cornea is similar to that which is seen after prolonged cocaine anesthesia with exposure of the cornea.

The question of whether the punctate relucant foci appear before the epithelial droplets (edema) is still unsettled. In some instances edema seems to be the first sign; in others, the relucant foci develop initially.

In untreated cases the corneal changes progress until the entire cornea becomes opaque. At this stage the surface is covered with fine stipplings and erosions. In optic section, opacification is most marked in Bowman's zone. In some instances the surface becomes entirely denuded of epithelium, except for a marginal rim at the limbus. Staining with fluorescein sharply outlines the denuded areas,

the edges of which may be raised and relucient. Deeper staining may occur under the edges.

In a case which illustrated this staining feature, tarsorrhaphy was



FIG. 223. Keratitis neuroparalytica seen through tarsorrhaphy opening, showing parenchymal changes in the cornea and vascularization.

performed; some time later, examination by optic section through one of the residual eyelid openings revealed that although the vascularized cornea was entirely opaque, owing to an intense scarring in Bowman's zone, the epithelium had regenerated, as indicated by a dark zone beneath an uninterrupted film line (Fig. 223).

If secondary infection supervenes, rapid corneal ulceration and breakdown may ensue with serious sequelae.

Filamentary Keratitis (*Keratitis Filamentosa*, *Keratitis Filiformis*, *Fädchen Keratitis*). In this condition epithelial filaments which vary in length and shape are formed on the corneal surface. The etiology is still unknown. Filament formation in itself does not seem to be specifically related to any one condition as it is observed in a number of corneal affections, such as recurrent erosions, herpes corneae, and keratoconjunctivitis sicca (Sjögren). In the latter, deficiency of tear secretion is known to be a concomitant factor. It may be that in certain instances the epithelial proliferations represent an ab-

normal repair process. Be that as it may, the idiopathic development of these epithelial filaments delimits filamentary keratitis as a definite clinical entity.

Filamentary keratitis is often confused with keratoconjunctivitis sicca, but in the latter condition elongated well-formed epithelial filaments rarely develop. Rather, one sees numerous flaky muco-epithelial accretions adherent to the corneal surface, which dull its luster. Sjögren believes that the corneal lesions in keratoconjunctivitis sicca (page 470) are part of a definite symptom-complex.

Corneal filament formation is nearly always associated with conjunctivitis but only rarely do filaments form on the conjunctival epithelium. The filaments consist of twisted, elongated epithelial cells admixed with mucus; they appear as knob-topped strands, very much like a miniature twisted umbilical cord (Plate XXXIV, fig. 5). The free bulbous extremity is dependent and is moved about by eyelid action.

In the early stages of development a whitish spot appears on the corneal surface; this forms the base of the proliferating filament. However, in some instances, intermediate forms are seen, in which rounded raised masses are present. There is a tendency for the longer filaments to be attached to the upper portions of the cornea. Rarely are more than ten separate filaments present. They may be visible to the unaided eye, but when they are small the biomicroscope will always reveal the true filamentary nature of these structures; characteristically they have a twisted, umbilicoid stem and a knobbed distal end.

The filaments are readily stained green with fluorescein. Complete development may occur in from twenty-four to forty-eight hours. As the filament elongates, movements of the eyelids cause it to twist, while admixture with mucus gives it a grayish and opaque appearance. It may become detached spontaneously and a new filament may arise and continue to grow on the same site.

The condition is characterized by remissions and relapses, and at times it becomes chronic. Frequently, there is a concomitant low-grade mucocatarrhal conjunctivitis. During the period of activity

there may be considerable irritation and photophobia as corneal sensitivity is rarely impaired.

INFLAMMATION OF THE DEEP ZONE (DEEP STROMA, DESCEMET'S ZONE, AND ENDOTHELIUM)

PARENCHYMATOUS KERATITIS (INTERSTITIAL KERATITIS)

Hereditary syphilis is the principal cause of parenchymatous keratitis, commonly known as interstitial keratitis.* However, statistics show that in *acquired syphilis* the incidence of this type of keratitis is only from 3 to 8 per cent. Therefore, typical interstitial keratitis should be thought of as a manifestation of hereditary syphilis.

The disease is a bilateral inflammation, characterized by edema, infiltration, and vascularization of the deeper corneal layers. It tends to run a chronic course and is almost always accompanied by uveitis. Hence, the disease must be considered as an involvement of the anterior ocular segment rather than of the cornea alone. It occurs twice as frequently in women as in men and it is most prevalent in the latter parts of the first and second decades of life. The aged are seldom affected. Bilateral ocular involvement is the rule but simultaneous affection of both eyes is rare. Involvement of the second eye occurs after an interval varying from a few weeks to one or more years. Spicer,²⁷⁹ in analyzing a series of cases, reported that in only 2 per cent of his cases, five years or more elapsed before involvement of the fellow eye.

As originally described by Hutchinson,¹⁵⁶ hereditary interstitial keratitis is usually associated with other stigmata of congenital syphilis. Among these are deafness, saddle-nose, peribuccal rhagades, dental dystrophies, arthropathies, socratic facies.

Biomicroscopic observation is of particular importance in the diagnosis of this condition, not only because it permits detection of the earliest signs of the disease but also because, even in healed

* Keratitis anaphylactica (Wessely⁸³¹), a condition clinically similar to interstitial keratitis of congenital syphilis, is due to allergy. Numerous cases have been reported in which interstitial keratitis was definitely associated with allergic states, particularly in individuals sensitive to certain articles in their diet. Elimination of the offending foods resulted in subsidence or even in complete recovery.

cases, characteristic diagnostic signs may be seen many years after the disease has become quiescent. The disease begins with changes in the endothelium and in Descemet's zone. These changes may



FIG. 224. Early stage of interstitial keratitis; little gray nebulae in deep stroma before vascularization.

precede the onset of subjective symptoms and of pericorneal injection by a week or more. They consist of a fine droplike diffuse sectorial or tonguelike opacification of the region of Descemet's membrane, caused by endothelial edema and other disturbances in the endothelial cells, which as a result appear bosselated. In most cases these changes start from the periphery at the upper part of the cornea but any portion of the cornea is vulnerable. This is followed by an infiltration in the form of small discrete faint gray spots, indistinctly outlined, in the middle layers of the cornea (Fig. 224).

Then, further symptoms (e.g., blurring of vision, photophobia), accompanied by pericorneal injection and corneal vascularization, appear (Fig. 225; Plate XXXV, figs. 1, 2). The spots (or maculae) increase in number and coalesce, resulting in a generalized haze which tends to progress toward the center of the cornea, where it usually meets and becomes confluent with processes extending from other sectors, until the entire cornea becomes cloudy, taking on a ground-glass appearance. However, in optic section the stromal infiltration is seldom solid. Small dark spaces (water-clefts) and irregular lines and areas of varying increased relucency are seen in front of the denser infiltrated and yellowish posterior face (Fig. 226).

In some cases, discrete maculae cannot be distinguished but if present, the use of sclerotic scatter aids in observing them. During this period further changes occur on the endothelial face; these may be seen in specular reflection, before they become obscured by the haze. The endothelial cells become polymorphous and more reflective. They lose their clear definition and become sievelike in appearance, with dark circular crevices between the cells, similar to the picture in endothelial dystrophy. At an early stage of the disease the involved areas show an increase in corneal thickness, which is manifested by a bulging of the posterior part of the optic section toward the anterior chamber.

The surface epithelium usually shows lack of luster, which is caused by epithelial edema and which may advance to the point of vesicle formation, and in extreme cases, to bullae. As the edema progresses in the deeper layers, characteristic folding of Descemet's membrane results, with the appearance of striate lines which have double reflexes and tend to form a criss-cross pattern.

At the same time keratic precipitates appear. They vary from small dots to large plastic masses or plaques. The deposits are usually found behind the involved sector, but they may be distributed as an inverted (V) pyramid at the lower part of the cornea, as they are in nonspecific uveitides. These deposits vary in color from gray to yellow or dark brown. At times they may be adherent to fine

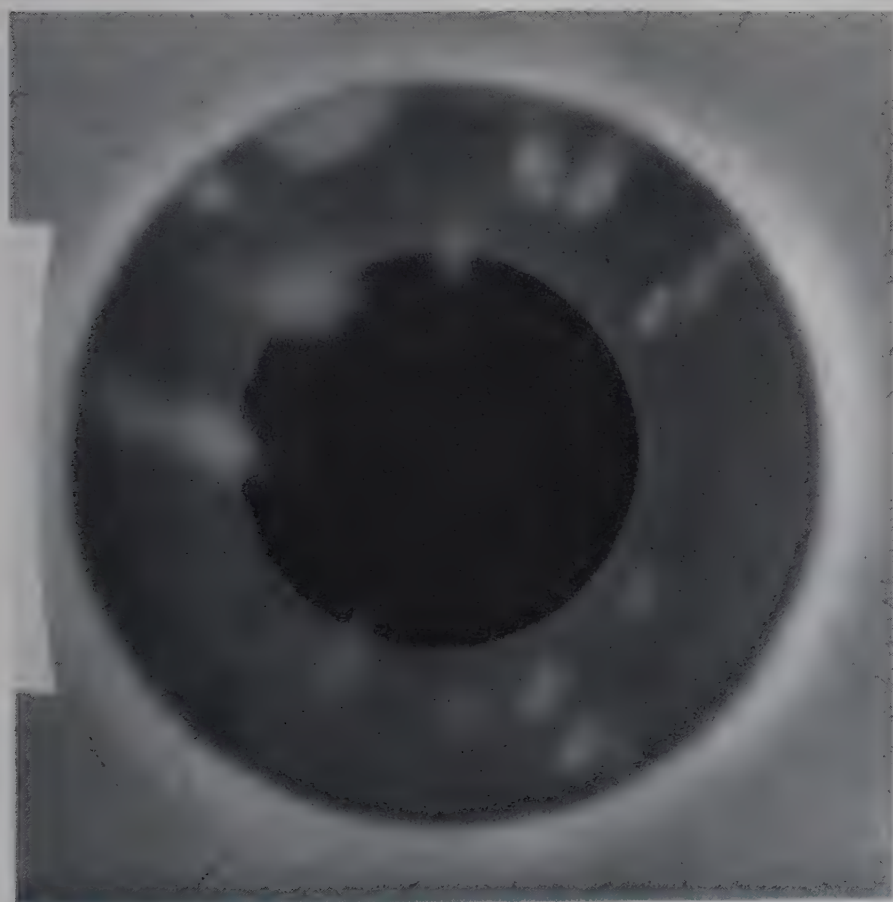
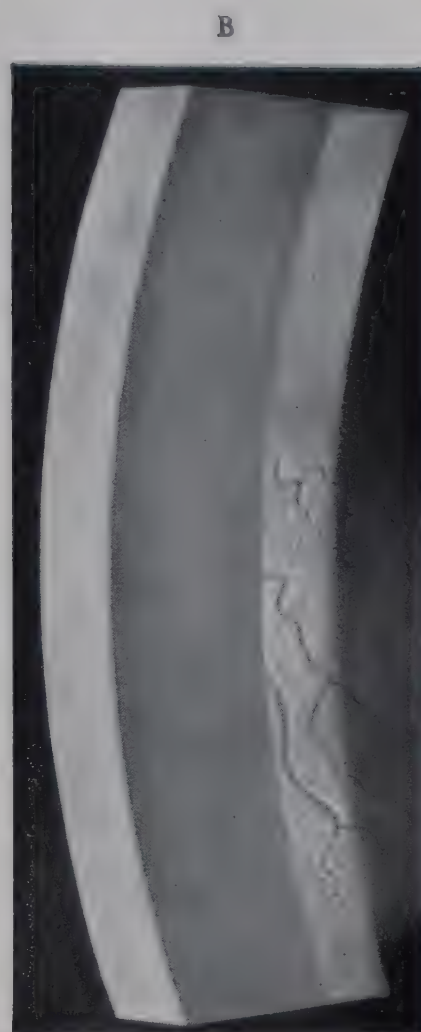


FIG. 225. A small nebula in the deep stroma of the left eye. A. Sclerotic scatter. B. The same lesion in direct focal illumination, showing beginning vascularization. C. Simultaneous appearance of lesions in the right eye showing a more advanced involvement of the stroma. Sclerotic scatter. D. Lesions shown in C by direct focal illumination. Note the posterior bulging of deep corneal face.

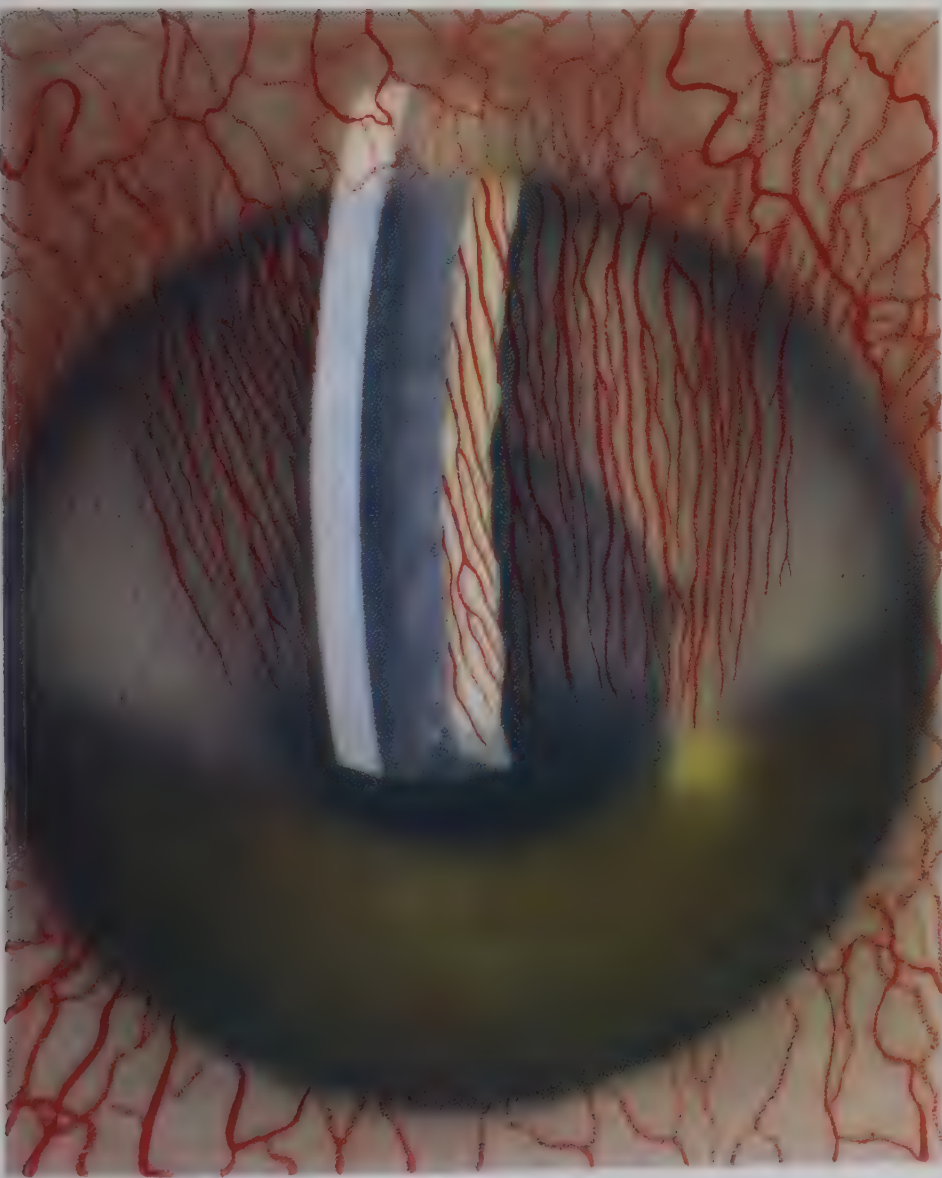
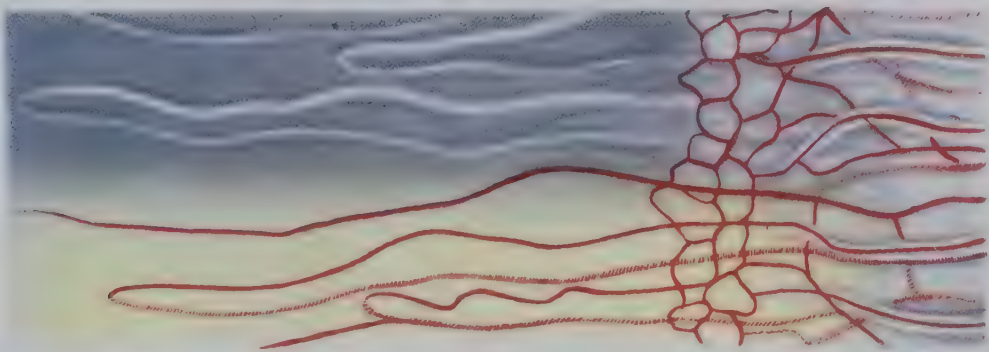
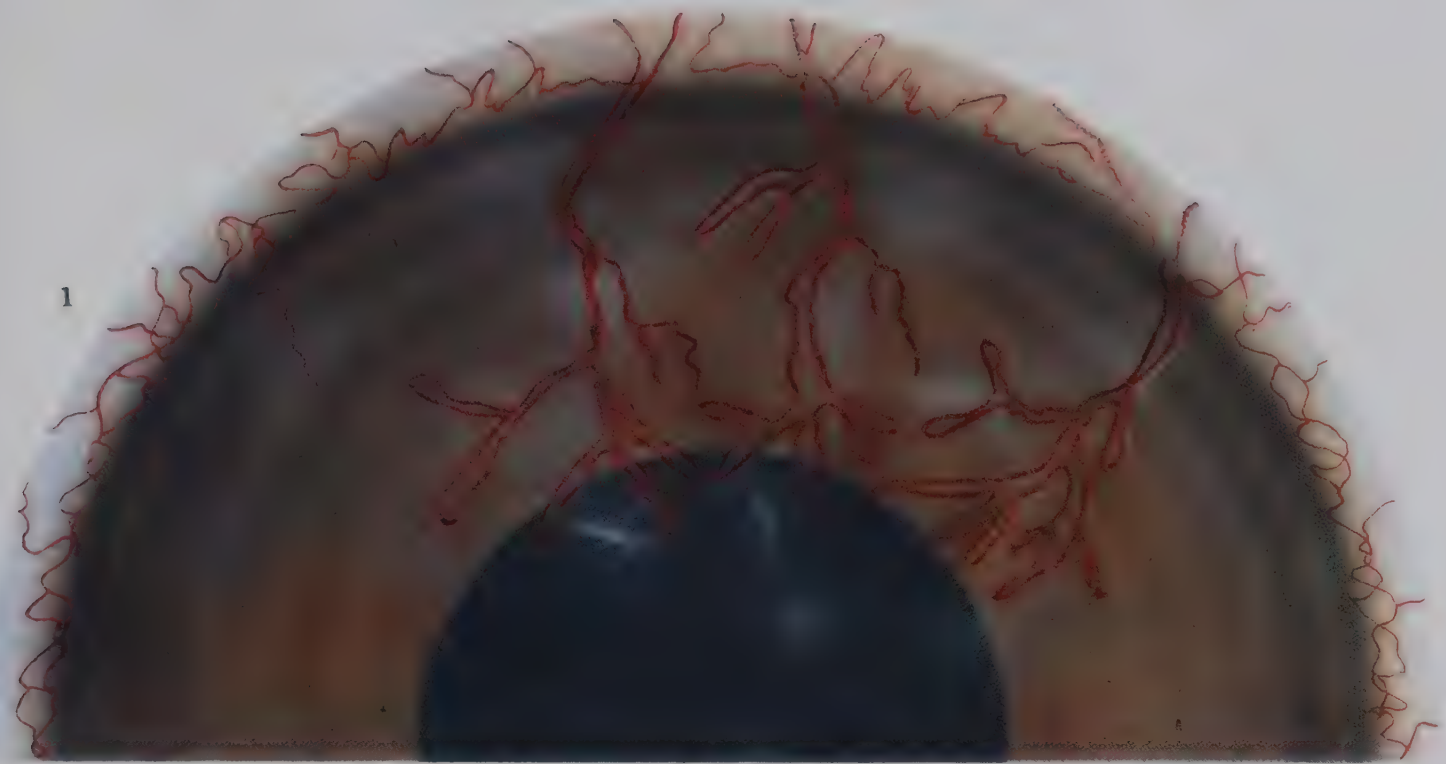
PLATE XXXV

FIG. 1. Early stage of interstitial keratitis showing nebulae in stroma and deep umbel type of vascularization. Diffuse illumination.

FIG. 2. Same lesion as Figure 1, in optic section.

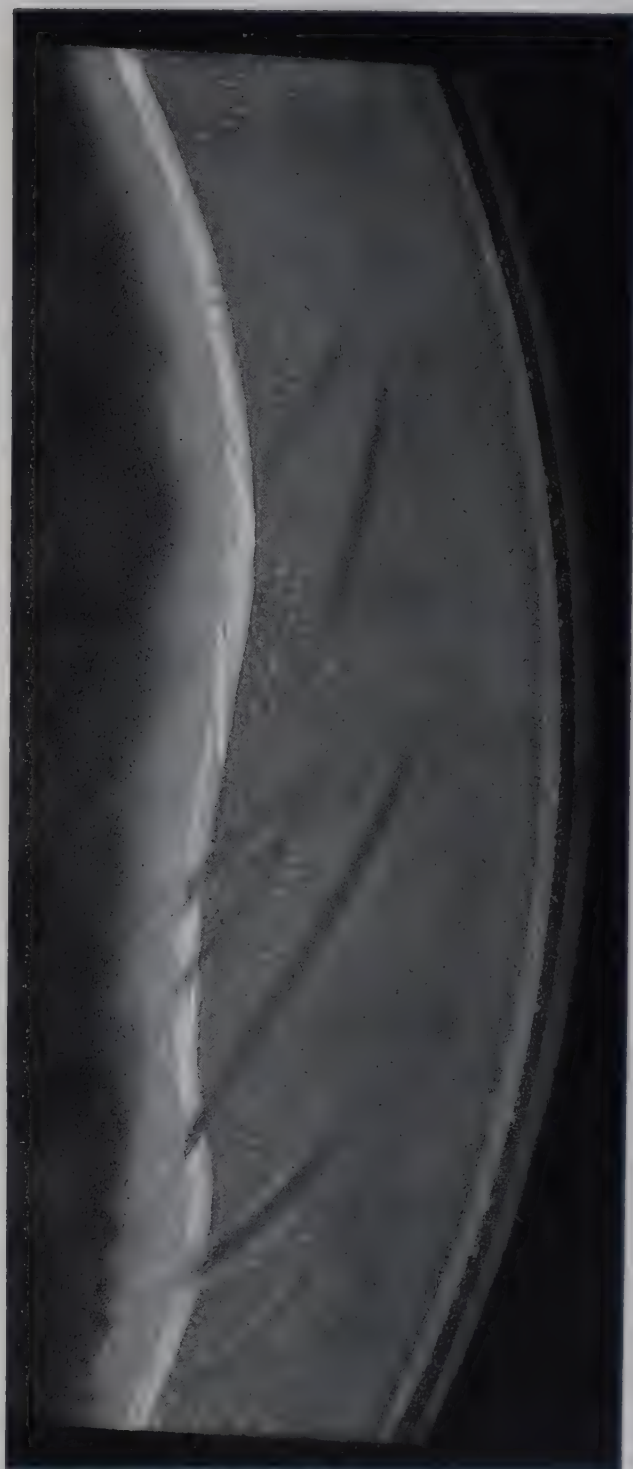
FIG. 3. Vascularization in acute interstitial keratitis, infiltration in the deeper parenchyma, especially in Descemet's zone. The salmon patch appearance is caused by a dense netlike vascularization in this zone.

FIG. 4. Interstitial keratitis. Neovascularization at limbus by direct focal illumination (above) and by retro-illumination (below). High power.





A



B

FIG. 226. A. Early stage of interstitial keratitis showing thickened and infiltrated cornea in optic section. In the diffuse haze are small maculae and tissue clefts. There is a marked relucency of the deeper zone and keratic precipitates. B. The same case two weeks later showing marked increase in relucency in posterior zones and loss of sharp outline of the posterior corneal surface due to infiltration and keratic precipitates (plaque-like).

fibrinous filaments attached to the endothelial surface. The presence of these adherent exudates in addition to the deep corneal infiltration gives a dirty yellowish irregular appearance to the deeper part of the optic section. This tends not only to obscure the earlier folds but also to make sharp outlining of the deep edge of the section difficult.

A trabecular network may be formed by the adhesion of fibrinous material to the summits of the folds on the posterior face of the cornea. Actually, these trabeculae are formed by strings of fibrin, agglutinated to the surfaces of the folded endothelium. The fibrinous exudates on the posterior face of the cornea may leave hyaline or glasslike membranes attached to the endothelium by one or both ends (Fig. 202). The effect of such changes tends to damage the endothelium permanently and together with the deeper corneal changes accounts for the typical increase in relucency of Descemet's zone, which is seen in old healed cases.

A more localized process (deposit of plastic exudates) on the posterior face in the form of a ring in the central portions of the cornea results in a form of interstitial keratitis known as keratitis centralis annularis of Vossius.

The initial corneal opacification is soon followed by a vascular invasion, which is the first sign of the development of a defense mechanism (Fig. 225 B; Plate XXXV, figs. 1, 2). The vessels follow the general direction of the infiltration. When the infiltration is sectorial, the vessels are usually limited to the involved sector at the onset but vessels may invade the cornea from any or all portions of its circumference. The latter type may occur at an early stage in the rare form of interstitial keratitis in which the lesion starts centrally.

Two types of vascularization occur: superficial and deep. The *superficial* vessels come from the terminal arcades of the conjunctival vasculature and run for a short distance under the epithelium, which may be raised to form a crescentic elevation at the corneal margin known as an "epaulet." These subepithelial capillaries are usually smaller than the deeper vessels. In other instances isolated

subepithelial loops may project from limbal arcades for a short distance at irregular intervals around the cornea.

The *deeper* vessels are derived from the anterior perforating ciliary vessels; their entrance into the depths of the cornea is hidden by the episcleral tissue. These vessels invade the deepest corneal layers just in front of Descemet's membrane in a radial manner, running centripetally and close together; they are brushlike and vary in size from small to fairly large. Such a formation, viewed through the hazy cornea, has a cherry red hue and is known as a "*salmon-patch*" (Hutchinson) (Plate XXXV, fig. 3). It is probably seen more often in the upper portions of the cornea than elsewhere. In other cases, in which the inflammatory reaction is not so marked, smaller capillary branches may course or loop irregularly through the deep layers of the stroma (Plate XXXV, fig. 1).

In most cases the deeper brushlike vascular invasion is preceded by a dense haze (more centrally). As the condition progresses toward healing, there is a tendency for the older peripheral infiltrates to become absorbed, leaving these areas relatively clear.

The processes of resorption or clearing are slow, depending on the density of the infiltration and vascularization. The first areas to become clear are usually situated in the more superficial layers of the cornea toward the periphery. Clear irregular stria appear in the opacified areas and tend to become more and more numerous. At first they are separated by opalescent infiltrated areas, which in turn may clear entirely. However, as a rule some opacification remains permanently as a diffuse deep nebular haze or in the form of scattered nebulae. Varying degrees of corneal thinning and ectasia inevitably follow. The superficial vessels tend to disappear. In practically all cases a permanent and diagnostic feature is the residual increased relucency of the posterior face (Descemet's zone), which is crossed by a dark capillary network, seen through the now clearer anterior layers (Fig. 227) (Plate XXXVI, fig. 1).

Soon after the acute phases of the disease have subsided, biomicroscopic examination reveals this characteristic vascularization, in which the granular corpuscular circulation may persist. These

vessels have a yellowish tinge in retro-illumination. In direct focal illumination they are seen as fine white lines. When followed centrally they are seen to turn back toward the limbus in "loop fashion."



FIG. 227. Interstitial keratitis — healed stage showing sclerosed relucet Descemet's zone and typical residual vascularization in the cornea. Chronic iritis.

The larger radial vessels (salmon-patch) become attenuated but also leave permanent evidence of their former presence. Lacking dichotomy, vessels can be differentiated from corneal nerves. However, by means of retro-illumination vessels may invariably be differentiated from all other structures. In the chronic or healed stages the presence of deep-lying capillaries on a relucet or fairly opaque whitish posterior face, associated with scattered parenchymal nebulae, is of especial significance in establishing the diagnosis of heredo-interstitial keratitis.

Atypical Forms of Interstitial Keratitis. Besides the typical forms already described, in which the initial stage of infiltration lasts a week or more; the florid or vascular stage, from two to six weeks; and the period of resorption, a year or more, the disease may appear in atypical forms.

Syphilitic Diffuse Interstitial Keratitis. This form, which occurs in acquired syphilis, is usually unilateral. While it may resemble the picture of heredospecific syphilitic interstitial keratitis of Hutchinson, it tends to evolve rapidly, being attended by only mild and attenuated reactive symptoms. It is manifested as a granular conglomeration of whitish yellow spots, which lie deeply in Descemet's zone. These regress rapidly with antisyphilitic therapy in contrast to the congenital syphilitic type, which does not respond rapidly to this treatment. In a more circumscribed type, though the infiltration may invade a considerable portion of the cornea, it rarely reaches the central areas. However, Spicer reported fulminating cases in acquired syphilis, in which the entire cornea became involved with the development of hypopyon. Mauthner and Hook²¹⁴ described a punctate type, which appeared during the secondary stage of acquired syphilis; numerous opacities were situated in the deep portions of the corneal parenchyma, but there were no other lesions of the eye. This is considered to be a "forme fruste," which heals rapidly without sequelae.

Avascular Interstitial Keratitis. This subacute variety, which seems to be characterized by lack of vascularization, has been known as an avascular type. However, examination with the biomicroscope late in the course of the disease may reveal isolated capillary extensions in the deeper layers. In other words, vascularization is minimal and delayed. A case of this type came to my attention. The patient was a man, 24 years of age, whose sole complaint was slight blurring of the vision of his right eye. His Wassermann complement fixation was weakly positive. There was no pericorneal injection throughout the course of the disease. In the region of the nasal limbus there was a tonguelike nebula, which in optic section was seen to be situated at the level of Descemet's membrane. The infiltration finally became granular in appearance. There were several delicate short folds in Descemet's membrane but only in the involved area. Optic section revealed a distinct localized thickening of the cornea (posterior bulging). Six weeks later, three or four capillary loops were seen to invade the sector from the periphery. They were situated just in front of Descemet's membrane and con-

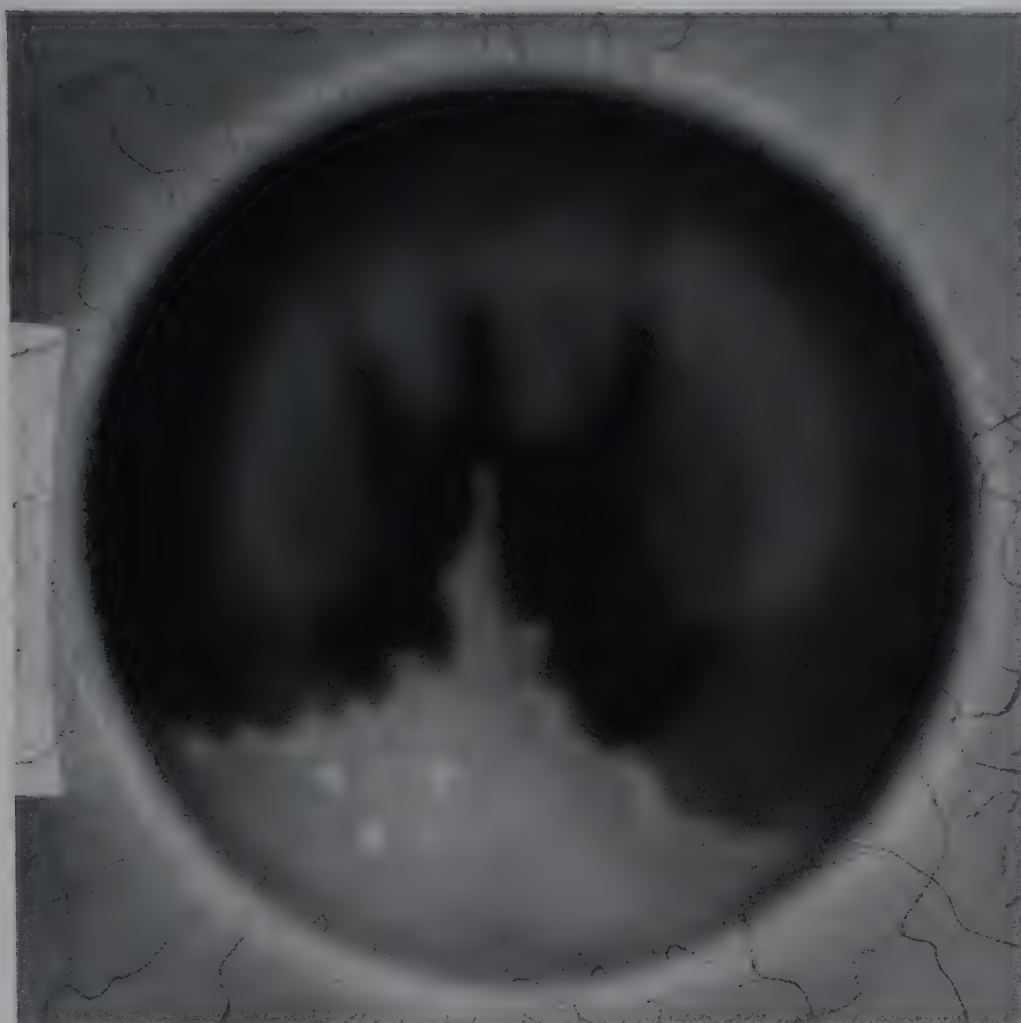
fined to one plane. The infiltration slowly progressed toward the center of the cornea but never actually reached it. After six months



FIG. 228. Avascular type of interstitial keratitis seen by sclerotic scatter.

the lesion became stationary with little resorptive healing except for a decrease in corneal thickening.

Another case occurred in a young woman who was in the fourth month of pregnancy (Fig. 228), and undergoing vigorous anti-syphilitic therapy. Her first symptom was blurring of vision, caused by a deep semilunar sector-like hazy infiltration, deeply localized, in the upper part of the cornea. The eye was white, but there was no evidence of iritis or aqueous flare. There were no corneal vessels. Eight weeks later two very fine capillary loops were seen invading the stroma. At this time there was no extension or change in the character of the infiltration. The right eye was unaffected. Four months after delivery (nine months after the original attack) she suddenly had an exacerbation of the keratitis in the form of a large triangular lesion in the lower part of the cornea (Fig. 229).



A



B

FIG. 229. A. Exacerbation of keratitis in lower part of cornea. (Same case shown in Fig. 228) nine months after the original attack (sclerotic scatter). The older healed lesions are still faintly seen above. B. Optic section through the margin of the new lesion showing thickening of cornea, stroma, deep involvement and a few keratic precipitates.

Other Types of Atypical Interstitial Keratitis. In one atypical type of interstitial keratitis the changes may be limited to the periphery of the cornea, the site of involvement corresponding to that occupied by an arcus senilis. In this case the infiltration is deep and may occur as a continuous circumferential band or in small semi-lunar areas. Sclerotic scatter reveals the shape and extent of such infiltrations. Although there appears to be a clear zone between the involved area and the limbus, optic section reveals relucant alterations and vessels in Descemet's zone, extending into the limbus. In other words, as in arcus senilis, the clear area is clear in its superficial layers only. The duration of this disease may be short and its course benign. On the other hand, there may be discrete foci which are unrelated to one another, each having its own independent vascular system.

In another form of keratitis the changes which are found chiefly in the posterior corneal face are characterized by keratic precipitation in the form of large fibrinous plaques. This type may result in the formation of a hazy ring around the center of the cornea, the so-called *keratitis parenchymatosa annularis of Vossius*.

Abscess formation on the posterior corneal surface or ulceration of the epithelial surface is rare. Fuchs has described a condition of deep pustular keratitis associated with severe anterior uveitis with acquired syphilis of long standing; this is characterized by nodular masses of a dirty yellow color in the deep corneal layers.

Keratitis Punctata Profunda (Mauthner²¹³). This is another variant of syphilitic corneal disease which is rarely seen today. It is distinguished by the occurrence of a number of discoid and punctate opacities in an otherwise unaffected cornea in association with syphilitic iritis. The lesions are grayish, somewhat discrete and well defined. They are confined mainly to the deeper parenchyma but they may become superficial. No vascularization occurs.

Sequelae of Interstitial Keratitis. Interstitial keratitis may have numerous sequelae. Chief among these are the various forms of damage which the cornea may suffer. Particularly prominent is the characteristic increased relucency of Descemet's zone and the ac-

companying residual vascularization. In practically all cases, areas of nebular haze (scars) remain in the deeper layers. The summation of all these forms of opacification leads to varying degrees of reduced

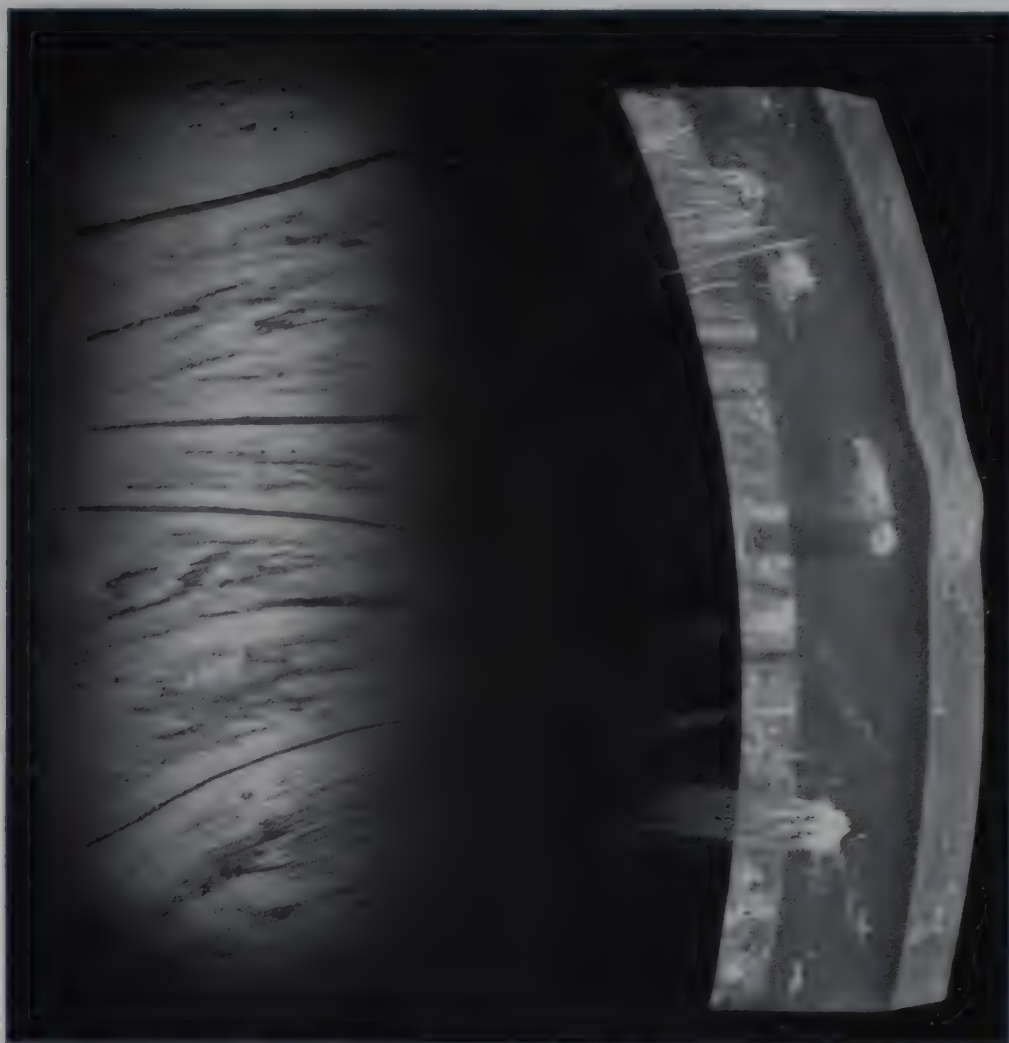


FIG. 230. Interstitial keratitis fifteen years following an acute attack. Optic section showing unusual feature in the stroma, possibly calcareous, and vascularization and increased relucency of Descemet's zone. To the left vascularization is seen by retro-illumination.

visual acuity (Figs. 230 and 231). Other sequelae may result from ruptures in Descemet's membrane which lead to the formation of concentric ringlike opacities, especially in the Vossius type of keratitis centralis annularis.

When the reaction in the anterior chamber has been severe, organized tissue may be observed in the form of a network attached to the posterior corneal face. Although precipitates are usually absorbed, when they remain in contact with the endothelium for a long time, they excite localized reactions, which leave permanent areas of increased relucency in the deep layers of the cornea. In addition, the associated iridocyclitis may lead to the usual complica-

tions following inflammation of these tissues, such as posterior pupillary synechiae, anterior peripheral synechia, atrophy of the iris, and complicated cataract. In extreme cases these complications may

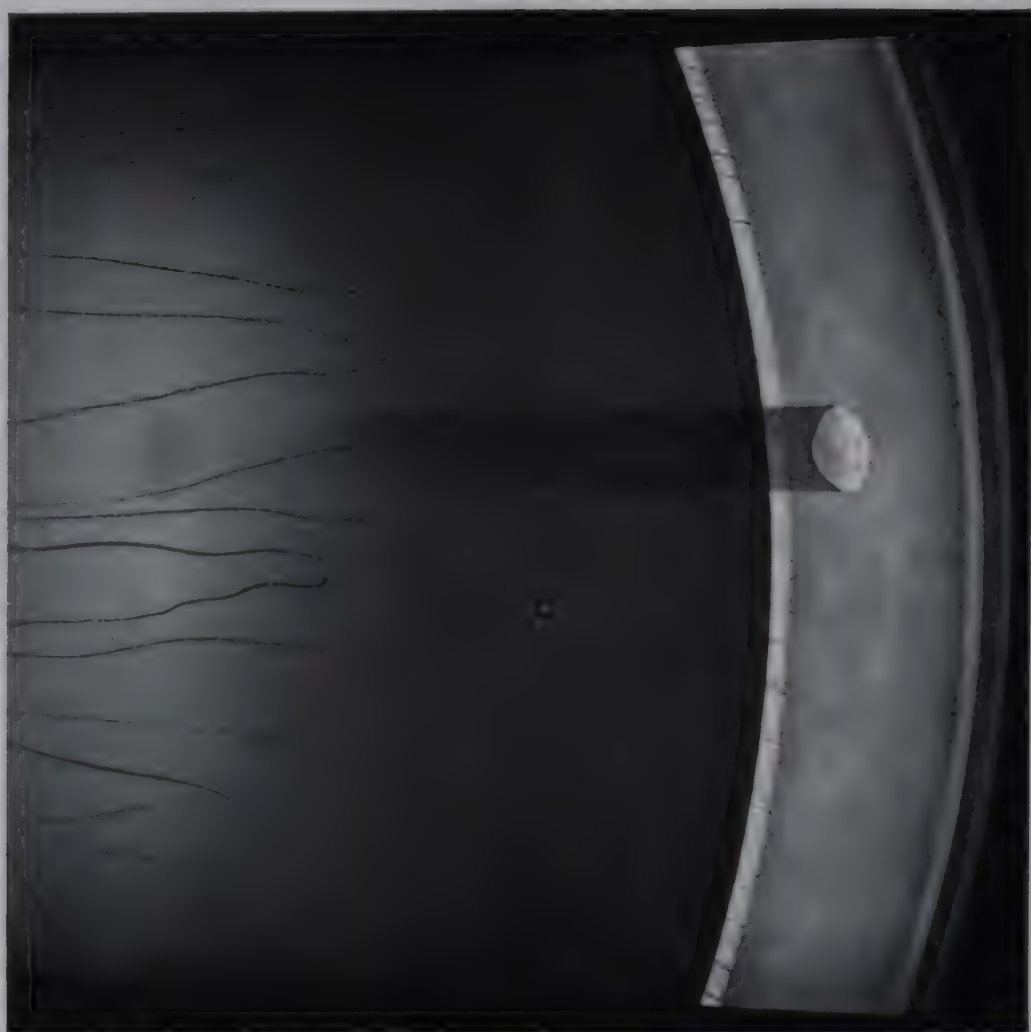


FIG. 231. Localized stromal scar, increased relucency, and vascularization of Descemet's zone resulting from interstitial keratitis for five years following an acute attack. Direct focal illumination. To the left, note vessels seen by retro-illumination.

cause secondary glaucoma and eventually atrophy of the globe.

Thinning of the cornea follows lamellar destruction and cicatrization. This change may be seen in optic section. It may be marked in extent, and lead to such weakening of the cornea that local or generalized ectasia results with consequent marked myopic and astigmatic refractive changes.

TUBERCULOSIS OF THE CORNEA

Tuberculosis affects the cornea in a variety of ways, which at times simulate other conditions, such as severe rosacea keratitis, or keratoconjunctivitis eczematosa. Consequently, there has been some confusion in the literature concerning the corneal conditions which

may be attributed to tuberculosis. Frequently, especially in the primary form, the diagnosis is made indirectly by exclusion or by the presence of other tuberculous foci or by proof of tuberculo-allergy (tuberculin reaction).

Corneal tuberculosis is usually a secondary manifestation of other tuberculous ocular disease. It may be manifested as a direct extension from the conjunctiva, sclera, or uveal tract, or as an allergic reaction.

Although a primary corneal type in which no other part of the eye seems to be involved has been described, it is doubtful whether such a type actually exists. Clinically, the primary type has been divided into infiltrated and ulcerated forms.

For the purposes of biomicroscopy the tuberculous lesions of the cornea may be classified according to the site of the involvement:

- I. Superficial
 - A. Extension from the conjunctiva
 - 1. Lupus (bacillary)
 - 2. Phlyctenular keratoconjunctivitis (tuberculo-allergic)
- II. Deep
 - A. Interstitial keratitis
 - 1. Bacillary with iritis
 - a. Via Schlemm's canal
 - b. Via aqueous
 - 2. Tuberculo-allergic
 - B. Sclerokeratitis and sclerosing keratitis
 - 1. Simultaneous presence of scleral and corneal disease
- III. Mixed types — combinations of any of the foregoing

It must be pointed out that this classification is essentially arbitrary since it may prove difficult to ascertain, even biomicroscopically (because of the almost constantly associated iritis), the exact route by which the cornea has become infected.

Lupus. The typical form of lupus of the cornea was described by Morax²²⁷ as an extension of a conjunctival or cutaneous lupus to the superficial layers of the cornea. Rarely, lupus of the conjunctiva and interstitial keratitis occur concomitantly. A pannus with grayish irregular separated nodules may develop which resembles

trachomatous pannus to a marked degree. The accompanying conjunctival lesions may also simulate trachoma because of the lupoid granulations present. Lupus is accompanied by superficial vascularization. The corneal epithelium is not interrupted but may become irregular and lusterless. The infiltration is densely grayish white in appearance (see lupus erythematosus conjunctivae).*

Tuberculous Interstitial Keratitis. Especially in the early stages, this condition may at times so closely resemble syphilitic interstitial keratitis as to be clinically indistinguishable on cursory examination. However, there are usually certain differences in the clinical picture of this disease which permits differentiation. In the syphilitic variety, although infiltration may start in one sector of the cornea it soon spreads, becoming a diffuse, "soft," deeply placed, grayish haze. In the tuberculous variety, however, the lesions in the beginning have a tendency to remain confined to one sector, usually in the inferior cornea. They soon become densely white in appearance. In the syphilitic type the infiltration is most intense in Descemet's zone where the maximum relucency is found, whereas in the tuberculous type the middle layers may be more densely affected. Also in the latter, intense nodular infiltrations may be observed within the gross lesion. Gallemaërts¹¹⁴ described these as roundish tubercles of a yellowish color within the middle layers.

The characteristic, deep, spearlike vessels of the "salmon-patch" of syphilitic interstitial keratitis are not seen in the tuberculous variety. In tuberculous interstitial keratitis, deep vascularization does not appear as early in the disease as it does in syphilis; it tends more to be confined to the superficial layers; the anastomosing arcades are much like the vascularization seen with the lesions of rosacea keratitis. The superficial vessels may be very large and tortuous.

The end results of tuberculous and syphilitic interstitial keratitis may differ markedly. The end result of tuberculous keratitis is usually a dense white leukomatous opacity traversed by enlarged, dilated superficial vessels, involving all the corneal layers and vary-

* Phlyctenular keratoconjunctivitis has been discussed on page 481.

ing in extent from one sector to the entire cornea. The typical residual picture of interstitial keratitis (luetica) is marked by a reluctant Descemet's zone traversed by a capillary network, while the clearer anterior layers may show soft irregular faintly gray nebulous areas.

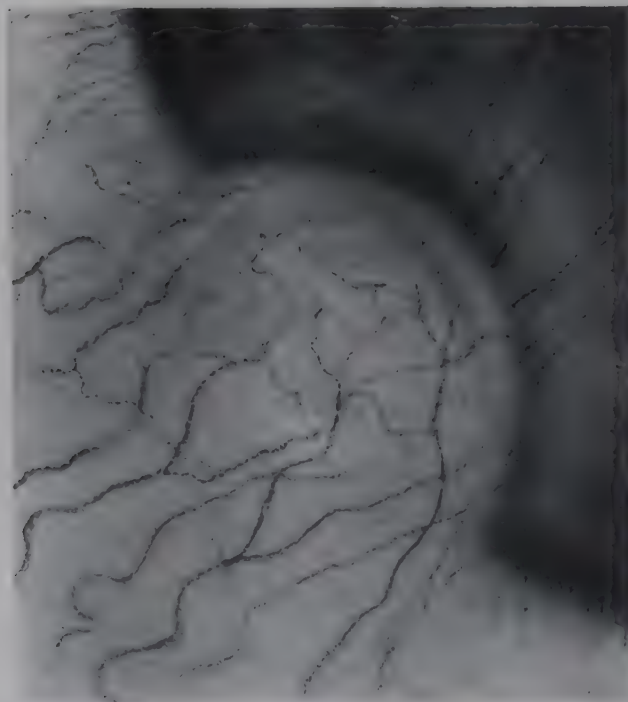
Sclerocorneal Tuberculosis. The extension of a tuberculous scleral infection into the cornea may give rise to a number of clinical pictures, depending on the severity of involvement. Although the tubercle bacillus has been implicated as the causal agent in these diseases, there is still some difference of opinion concerning this. Whatever name is applied to the condition it must be recognized that there is a concomitant infection of both sclera and cornea, and that the best designation is sclerocorneal tuberculosis.

Clinically, the diagnosis of the tuberculous nature of the ocular disease may rest on the finding of other tuberculous foci in the body. Sclerocorneal lesions must be differentiated from those of Boeck's sarcoid, rosacea keratitis, gouty episcleritis, syphilitic gummas, lepromas, and sarcoma of the limbal regions.

Sclerocorneal tuberculosis usually begins in the sclera, frequently in both sclera and cornea simultaneously. Some degree of iritis is usually present. The scleral onset may be either diffuse or nodular. In the former there is localized or fairly widespread episcleral congestion. With the narrow beam a thickened and congested episclera is revealed. The depth of the section will have a yellowish tinge. In the nodular form there is a little rounded, slightly swollen tumor, adherent to the sclera near the limbus. It may be quite hard and cartilaginous in consistency. The nodule is grayish in the center with a reddish yellow periphery. The narrow beam shows the elevated surface of the nodule, particularly when it is stained with fluorescein. Surrounding this nodule is a festooning of blood vessels. In the beginning the conjunctiva is freely movable, but later it becomes adherent and ulceration may ensue. Differential staining reveals the dark necrotic center vividly. The fate of these scleral lesions differs. They may resolve with varying degrees of scleral scarring; or following ulceration the contents of the nodule may evacuate and



A



B

FIG. 232. A. Tuberculous interstitial keratitis by sclerotic scatter showing old corneal scars and fresh nodule at the limbus. B. Nodule at the limbus (high power).

healing may leave a slaty, thinned-out scleral scar. On the other hand, deeper extension and caseation necrosis may result in a perforation of the sclera. Sometimes a scleral nodule is seen, somewhat removed from the limbus, without corneal involvement. The closer the scleral lesion is to the limbus the more likely is the cornea to become involved. When the cornea is affected a localized area of interstitial keratitis develops in a sector adjacent to the scleral nodule.

Distant corneal infiltration occasionally occurs. I have recently seen a case in a man, 35 years of age, who had tuberculosis of the bronchial lymph nodes. The Mantoux reaction was positive (Fig. 232). There was a raised vascularized limbal nodule, which gradually developed in the lower nasal quadrant. Adjacent to this there was a deep interstitial tonguelike infiltration, extending 2 mm. into the cornea with no intervening clear area. Above, in the superior cornea, a semilunar band of avascular infiltration extending into the deeper corneal layers was seen about 3 mm. from the upper limbus. There was no connection between the two lesions. Mild iritis was present. After two months the condition quieted and the scleral nodule flattened out. The adjacent infiltration was seen as a faint haze with two deep capillaries in the middle layers of the cornea. The superior nebulous haze was still present and was easily seen by sclerotic scatter. There was no vascularization discernible in this area.

Such a process in its graver form, occurring at the limbus, may give rise to a severe type of sclerokeratitis in which larger areas are involved with dense infiltration through all the corneal layers without any intervening clear area at the periphery. Direct continuity of the opaque sclera and the corneal infiltration (sclerosing keratitis) may thus develop. A lesion of this type is usually accompanied by deep and superficial vascularization, and a dense porcelain-like scar remains as a residuum. Localization to one portion of the cornea is common but extension circumlimbally may occur and eventually the whole cornea may appear porcelain-white (Fig. 233) and vascularized.

Similarly, under the designation *scleroperikeratitis*, von Szily³³²

described an even more acute malignant form which progresses rapidly over the cornea. Although gout and syphilis have been implicated as etiologic agents, current opinion favors tuberculosis. In the defi-

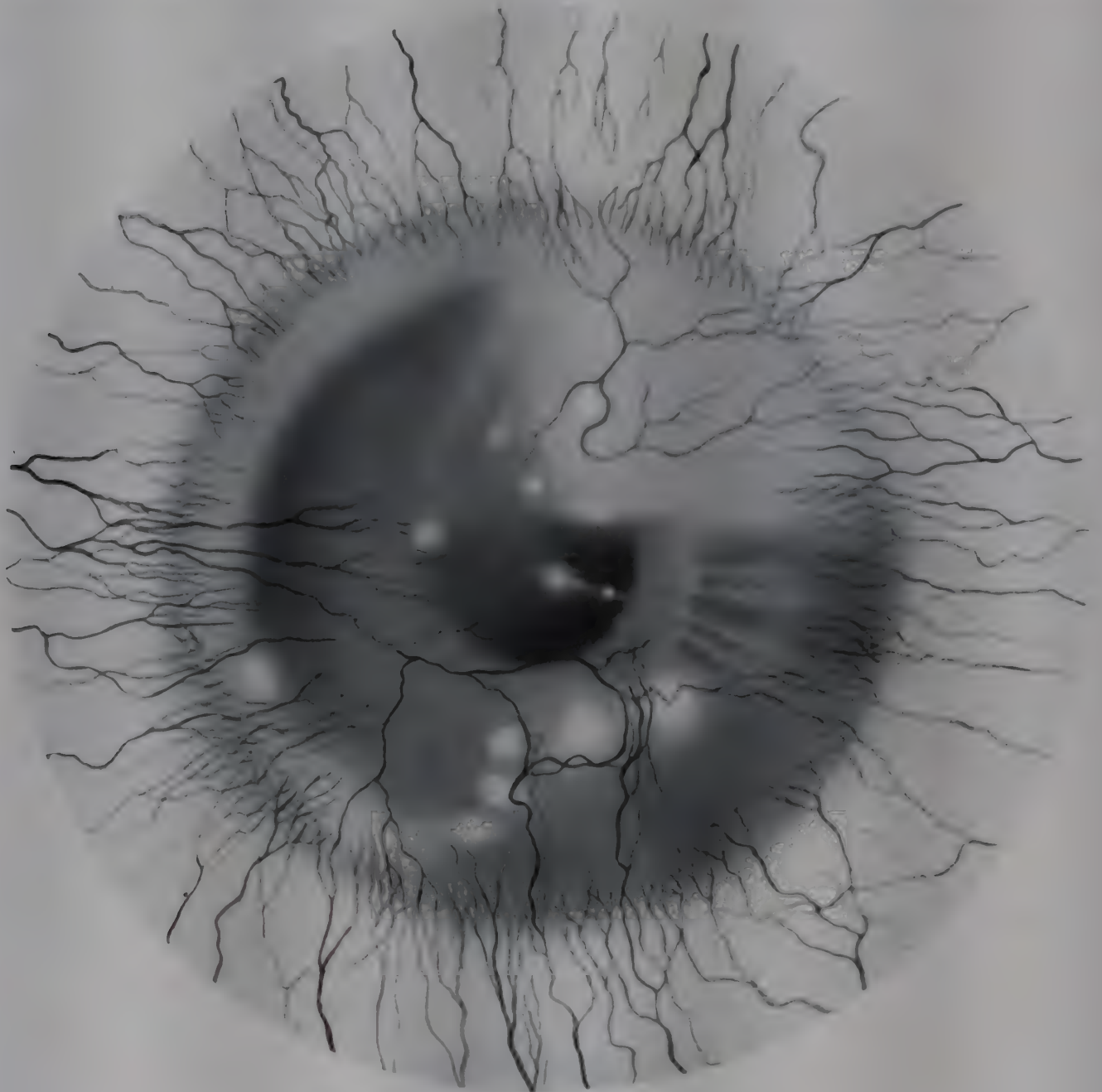


FIG. 233. Scleroperikeratitis tuberculosa.

nately tuberculous scleroperikeratitis there may be certain differentiating features. These are, first, the presence of whitish yellow nodules in the deeper parenchyma (tubercles) and second, the characteristic appearance of the scleral nodules.

Exacerbations and recurrences may occur in all forms of sclerokeratitis. Recurrences have been known to appear years after the initial attack. Although the first lesion may be minimal, later attacks may so spread the lesion as to constitute a form of progression.

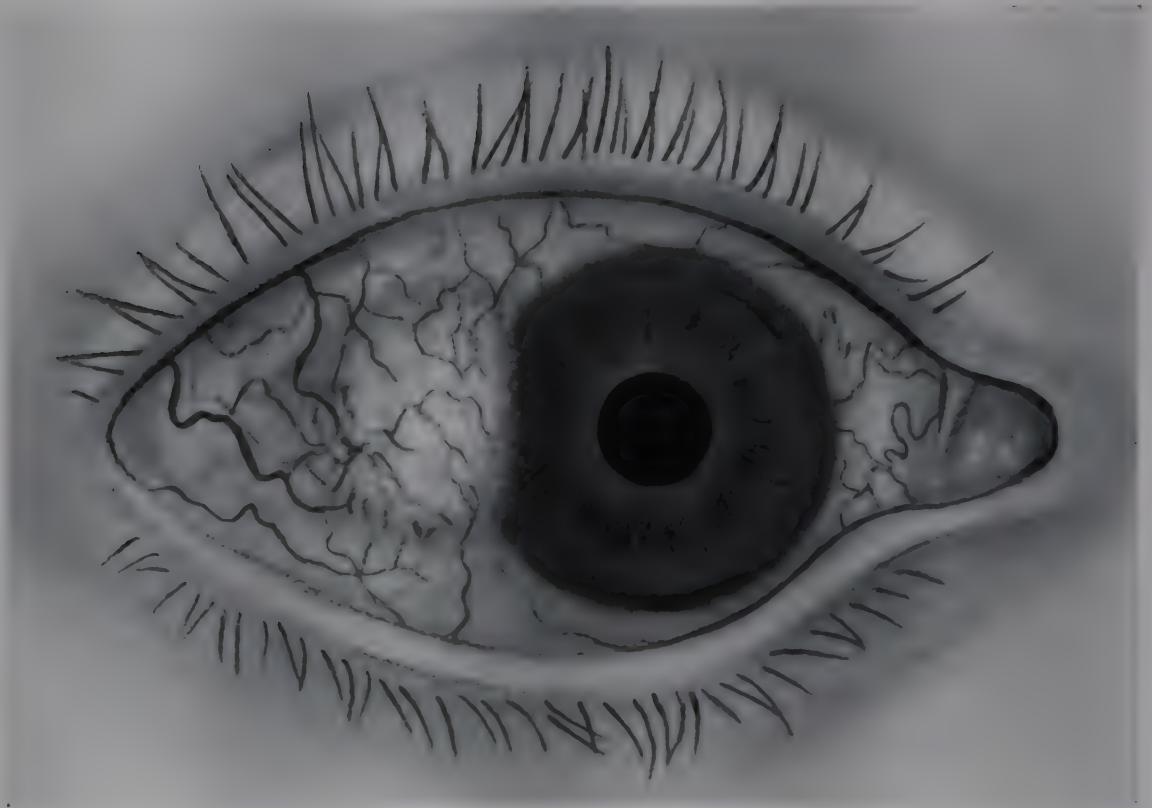
LEPROUS KERATITIS

Barros²² divided the corneal changes brought about by leprous keratitis into two principal groups: (1) hyperplastic keratitis and (2) infiltrative parenchymatous keratitis. In addition to keratitis e lagophthalmos, resulting from exposure of the cornea due to cicatricial eyelid damage or orbicularis paralysis, and corneal changes secondary to iridocyclitis, Barros also observed a case of band-shaped keratitis.

The hyperplastic type of leprous keratitis is the result of extension over the cornea of a leproma from neighboring sclerotic lesions, especially those which originate at the limbus. The limbus seems to be the preferred site for the hyperplastic or tumorous type of lesion, which at the outset appears as a small, yellowish, raised nodular excrescence producing no subjective discomfort. Extension over the cornea is slow and is accompanied by abundant vessels from the superficial and deep limbal arcades. The lesion has a yellow color and invades the deeper layers of the cornea, first producing vascular sclerokeratitis of a sclerosing type as the superficial layers become involved. There is marked corneal thickening with both anterior and posterior bulging. At times the lesion on the limbus and over the cornea as well, may develop into a large solid hornlike mass, which may interfere with closure of the eyelids. Central degeneration of this mass may occur with cyst formation. Necrosis with sloughing of the hyperplastic mass may ensue with ultimate formation of a corneal staphyloma.

Infiltrative parenchymatous keratitis may occur as an isolated corneal lesion (Fig. 235) or, rarely, in association with the hyperplastic type of leprous keratitis. In this form the infiltration appears as a superficial punctate keratitis or interstitial keratitis or as a leprous pannus.

The first biomicroscopic sign of infiltrative parenchymatous keratitis is the presence of fine white or golden dots mainly confined to the upper temporal cornea in the anterior layers. These dots may be found some distance from the limbus, usually in a sleeve-like arrange-



A



B

FIG. 234. Leprous keratitis. A. Sclerosing type of leprous sclerokeratitis (hyperplastic form).
B. Hyperplastic sclerokeratitis of leprosy, advanced stage.

ment around a corneal nerve fiber; or they may be found in the vicinity of the limbus in a wedge-shaped distribution with the base of the wedge upward. The juxtalimbal portion of the infiltration

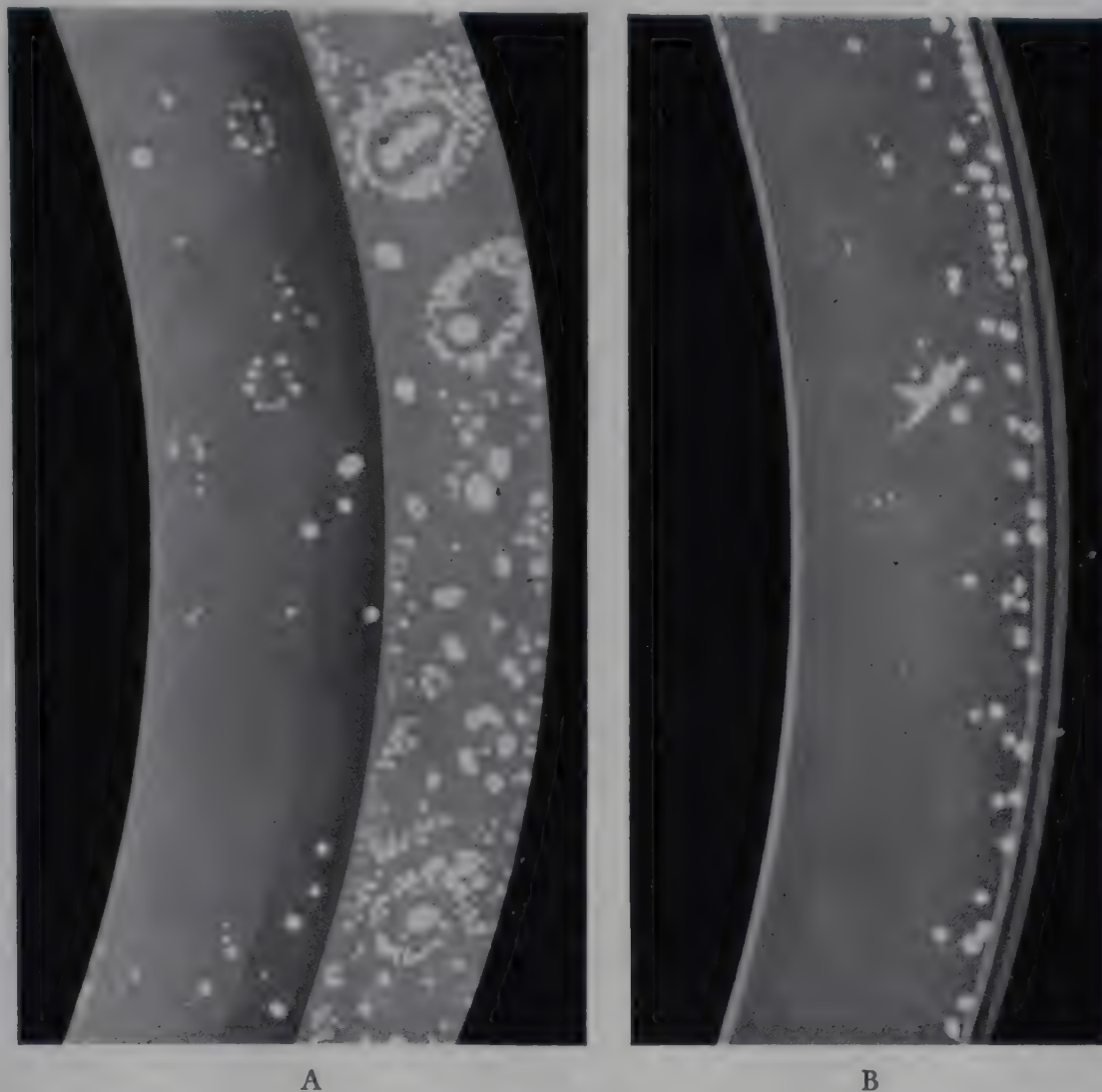


FIG. 235. A. The corneal lesions in the infiltrative parenchymatous form of leprous keratitis as seen by direct focal illumination. (After Barros.) B. Same lesions in optic section.

may extend into the deeper layers of the cornea, while the infiltration at the apex of the wedge is placed more superficially so that the optic section reveals a conelike distribution. The location of these infiltrates may vary. In some instances they are confined to the superficial layers of the cornea and, when large, may cause an elevation of the film line.

Barros found cases in which the infiltration was localized in the superficial and the deep layers, leaving the middle layers free. As the infiltration increases in intensity the picture resembles a grayish veil-like cloud interspersed with larger punctate opacities, which extends from the upper and temporal limbal regions.

The spread of the lesion may follow a fairly definite sequence; beginning in the upper temporal quadrant, it spreads to the upper nasal and lower temporal quadrants, and invades the lower nasal quadrant last.

Years elapse before the cornea becomes opaque, and there may be no vascularization with the exception of small peripheral vascular loops in the deeper layers. However, marked corneal edema associated with iridocyclitis may stimulate corneal vascularization, both superficial and deep, so that in the end a true pannus crassus may develop.

There is marked increase in visibility of the corneal nerves so that they may even be seen by retro-illumination or sclerotic scatter. This is probably due to the fine sleeve-like infiltrates surrounding them. Although corneal anesthesia has been mentioned as a prominent finding of leprosy, Barros does not agree with this, he states that he has not found alteration in corneal sensitivity even in advanced lesions. The tendency toward predominance of the corneal infiltration in the anterior portions of the cornea, especially at the onset in the early stages may be due to the neurotrophism of *B. leprae*.

With iridocyclitis there may be an increase in corneal opacification, due to the exudates on the posterior corneal surface and the endothelial and epithelial edema. Erosions and ulcerations are infrequent except in cases of paralytic lagophthalmos.

LEISHMANIA KERATITIS

Corneal inflammation may develop as a complication of Delhi or Aleppo boil. The disease is rarely seen outside the tropics. At the outset it appears as a localized infiltration not unlike phlyctenular keratoconjunctivitis. All the layers of the cornea may become involved very quickly leading to abscess formation and corneal perforation. The corneal lesion may result from extension of leishmania conjunctivitis.

KERATITIS PROFUNDA

This is a nonspecific type of parenchymatous interstitial keratitis occurring in adults. It is usually unilateral, more circumscribed,

and less severe than Hutchinson's type. It is generally accompanied by anterior uveitis. Both clinically and biomicroscopically its appearance so closely resembles the heredospecific form as to be indistinguishable from it. In some respects it is similar to the deeper corneal reaction which is seen in herpes zoster. It occurs more often in patients with lowered resistance — the aged, and those chronically ill, or suffering from toxic infections. Foci of infection and exposure to cold or mild trauma are believed to play a role in the etiology of this lesion.

KERATITIS DISCIFORMIS (FUCHS)

This indolent central keratitis is characterized by its discoid shape and occasional concentric configuration. It is considered to be caused by exogenous agents. There is usually a history of trauma. One modern conception of its etiology is that it is produced by a virus infection following an abrasion of the cornea. It begins with localized central epithelial edema of the cornea, which causes loss of luster and irregularity of the corneal surface, especially evident when seen in specular reflection. Rarely, in the earliest stages a stainable epithelial abrasion can be seen in the center of the discoid process; this may be the point of entrance of the infection. Following this, central opacification of the middle layers of the cornea develops; this opacity appears to be composed of closely packed minute grayish dots, surrounding relucet edematous areas having the so-called water-clefts (linear or fusiform dark streaks bordered by opaque edges). When such changes are marked, they may produce, in diffuse illumination, a streaked appearance, which at times gives the opacity an irregular stellate form. Such alterations, the effect of an intense parenchymal reaction, are seen in other acute interstitial involvements, for example, heredo-interstitial keratitis. The infiltration and edema tend to spread centrifugally, forming an enlarged disk, which rarely, if ever, reaches the limbus (Fig. 236). The margin of the central disk may be preceded by a hazy concentric ring of parenchymal edema and infiltration. Optic section discloses marked thickening of the cornea in the affected area, characterized

by bulging of the posterior face of the cornea toward the anterior chamber (Fig. 236 B). This corneal thickening may be of long duration. Examination with sclerotic scatter is most useful in disclosing the discoid nature of the lesion. As the condition progresses the epithelial edema may become less marked, leaving a clear, transparent, anteriorly situated layer of corneal tissue in front of the infiltrated scar (Fig. 237 A and B).

As a rule vascularization does not appear for from several months to a year after onset. Brushlike vessels resembling those of interstitial keratitis penetrate the cornea from the limbus and tend to migrate over the surface of the opacity, forming interanastomosing arcades. Folds in Descemet's membrane are present.

In mild avascular forms resorption occurs quickly but in most instances the opacity becomes organized into an opaque central scar of varying density. The residual capillaries remain permanently.

SUPPURATIVE KERATITIS — ULCERS OF THE CORNEA

Except in the early stages or during the period of regression and cicatrization, biomicroscopic examination of an infected ulcer may be extremely difficult. The accompanying symptoms (photophobia, lacrimation, and swelling of the eyelids) may be so severe as to make the examination intolerable and at times even inadvisable.

The conditions in which ulceration occurs as secondary complications from loss of epithelial vitality have already been discussed. These include a host of superficial keratitides, in which the epithelium becomes edematous and eventually eroded. Many of these ulcers are the result of the extension of conjunctival disease. Except in the rare cases complicated by secondary infection, such erosions or ulcers of the epithelium heal readily by means of regeneration of the epithelial cells, and leave few or no sequelae.

There is one other exception: when the ulceration involves Bowman's membrane, scarring results, even though there is no secondary pathogenic invader. It must be remembered that a secondary microbic infection may cause a severe suppurative ulcer that will overshadow the original condition.



A



B

FIG. 236. Keratitis disciformis (early stage). A. By sclerotic scatter. B. In optic section through the center of the lesion.



A



B

FIG. 237. Disciform keratitis (later stage). A. By sclerotic scatter. B. In optic section. (Courtesy of Dr. I. Givner.)

However, the usual infected ulcer follows an exogenous infection superimposed on a traumatic solution of continuity of the epithelium. Although the extent of the initial break in the epithelium may be almost imperceptible, the entrance of virulent micro-organisms may result in suppurative ulcers of varying severity. These vary from the small localized *catarrhal* or *Morax-Axenfeld* type of marginal ulcer, on the one hand, to the fulminating serpiginous ulcer (pneumococcus) with hypopyon, on the other.*

The initial biomicroscopic appearance varies, depending on whether there has been pre-existing infiltration or whether the ulcer has resulted from a simple abrasion of an otherwise normal corneal epithelium with secondary infection. In the latter case infection causes a reaction with swelling and cloudiness of the borders of the abrasion and consequent interference with normal epithelial repair. At this stage of development the floor and edges of the defect stain readily. As the loss of substance increases dazzling reflexes are produced by the surface irregularity. Infiltration about the ulcer results in the formation of a disk-shaped opacity which involves the anterior layers of the stroma. The epithelium over this hazy area becomes edematous. As the disease progresses, this edematous epithelium breaks down and the crater enlarges. At the same time, the deeper layers of the parenchyma become swollen and folds in Descemet's membrane may appear.

The sloughing floor of the ulcer is rough and opaquely gray; its edges are creamy yellow, and there is intense conjunctival injection. Further progress depends on the virulence of the infecting organism, resistance and therapy. Healing may occur at any stage of the infection. If this happens then the necrotic material disappears from the floor of the ulcer and its early roughness smooths out owing to epithelialization of the defect.

The amount of residual scarring depends on the amount of tissue

* It is not the purpose of this discussion to delve deeply into the manifold varieties of corneal ulcers, since most classifications deal largely with the etiologic, topographic or pathologic differences and do not concern themselves with biomicroscopic distinctions or details. Therefore, only the biomicroscopic appearance of corneal ulceration in general, with the addition of certain specific types which demand special consideration, will be described.

substance lost; if this is not marked, either a nebula or a macula results. Thinning of the cornea always occurs, but the ingrowth of epithelium may fill out the anterior defect, so that no surface irregularity remains. In such a case optic section shows that there is no interruption and little irregularity of the precorneal film line over the scar. Below this a dark underlying widened space represents the thickened epithelium.* Following severe ulcers, however, faceting of the surface due to scarring in Bowman's zone usually occurs (Fig. 239). If healing does not occur, further progression of the ulcer leads to enlargement of and increase in the severity of the alterations. In these cases the entire thickness of the cornea at the site of the lesion is affected. In some instances the process seems to start below the epithelium as an infiltration, the avenue of entrance in the epithelium not being apparent. This results in an abscess of the parenchyma, which may develop into a typical serpiginous ulcer. In the beginning superficial crater-like loss of substance is not seen. However, later the devitalized epithelium may become necrotic, producing a superficial loss of substance with a depressed, sloughing floor.

As a rule, the closer the ulcer is to the limbus the sooner vascularization occurs. This is usually a superficial extension from the pericorneal limbal vascular arcades, but in severe cases, deep vascularization may occur. The vessels enter the involved area and, at times, in the regressive stage, small irregular reddish areas may be observed. By means of direct focal illumination these areas are seen to be composed of congeries of capillaries. Limbal ulcers frequently heal with the formation of pseudopterygium of varying size. The swollen infiltrated conjunctiva encroaching over the lesion leaves a raised vascularized scar continuous with the conjunctiva.

Ulcus Serpens. In virulent *ulcus serpens* the process tends, either from the direct extension of the infection or from the toxins, to spread irregularly. This is indicated by an increase in epithelial edema, circumferential or on one side, in the area surrounding the ulcer. Radiating, spearlike or circular lines of infiltration, below the epithe-

* Regenerated epithelium is always nonrelucant and nonresponsive.

lial edema, may be seen in the deeper layers extending toward the periphery. There may also be an increase in central sloughing (Plate XXXVI, figs. 2, 3).



FIG. 238



FIG. 239

FIG. 238. Serpiginous ulcer of the cornea with hypopyon below. Sclerotic scatter.

FIG. 239. Optic section through the center of lesion shown in Figure 238. Note that the surface of the ulcer has been epithelialized and that the surface of Bowman's zone is irregular.

At this stage pericorneal injection increases and the associated iridic irritation tends to the formation of *hypopyon* (Fig. 238). In addition there may be extensive fibrinous deposit on the posterior surface of the cornea behind the ulcer. This may remain for a long period of time, even after regression and cicatrization have taken place. In the deeper layers, dense localized infiltrations (minute abscesses) may signify the development of a larger posterior abscess. Such deep reaction in the cornea may produce posterior bulging toward the anterior chamber. In these severe ulcers, perforation of the cornea may be beneficial and a forerunner of the regressive stage, provided such complications as incarceration of the iris or even pan-

ophthalmitis do not occur. However, in certain instances, even severe ulcers may heal without perforation.

If an ulcer regresses spontaneously and the denuded area becomes filled by epithelialization, the subepithelial opacity appears to consist of swirls of whitish bands and lines, spun out into an irregular mass. At this time, the film line (in optic section) is seen to be intact but slightly undulating, raised (bullous-like elevations) or depressed over the opacity. In some cases a thickened, infiltrated and bulging posterior corneal surface behind the ulcer may be demarcated from the neighboring uninvolvement cornea by a sharp line. This line represents the change in curvature from the normal posterior curve to the abrupt swollen and infiltrated posterior aspect of the ulcer. Treatment (cauterization) may modify the entire picture and cause a widely divergent appearance.

Cicatrization is always associated with thinning of the cornea (Fig. 240). The ultimate amount of scarring varies in individual cases. There is a tendency, however, for scars to clear in certain cases, especially in the young or when vascularization is marked. Dense whitish scars like those remaining after severe ulcers are known as leukomas. If perforation with iris incarceration has occurred, an adherent leukoma results. A thinned out corneal scar may become ectatic. In adherent leukoma, extensive ectasia may result in anterior staphyloma. (See discussion of scars, page 53.)

Marginal Ulcer. As previously mentioned, this type of ulcer is usually associated with a pre-existent conjunctival disease, which initiates a subepithelial infiltration near the limbus. At the onset, the infiltration consists of groups of small white dots just below the epithelium, which becomes raised. Coalescence occurs with the

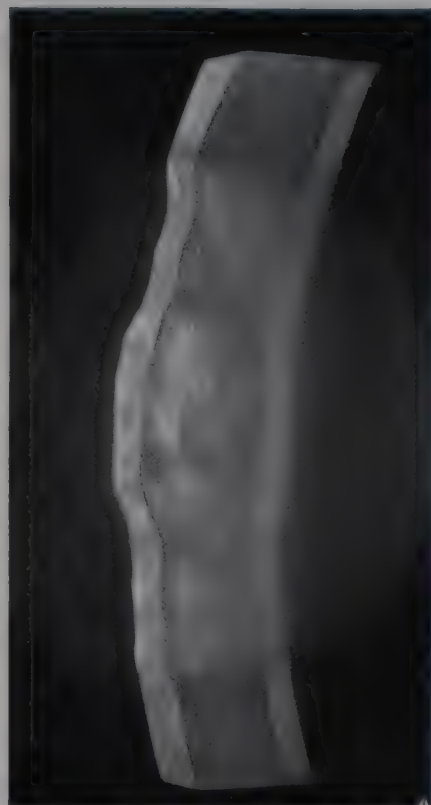


FIG. 240. Corneal scar following serpiginous ulcer of the cornea illustrating irregularity of the anterior corneal surface and thinning due to loss of substance.

PLATE XXXVI

FIG. 1. Quiescent stage of interstitial keratitis. The parallelepiped shows the residual relucency of Descemet's zone and typical vascularization of this layer. The vessels appear whitish and hence might be mistaken for nerves or folds. The vascular nature of these lines is revealed better to the right by direct retro-illumination.

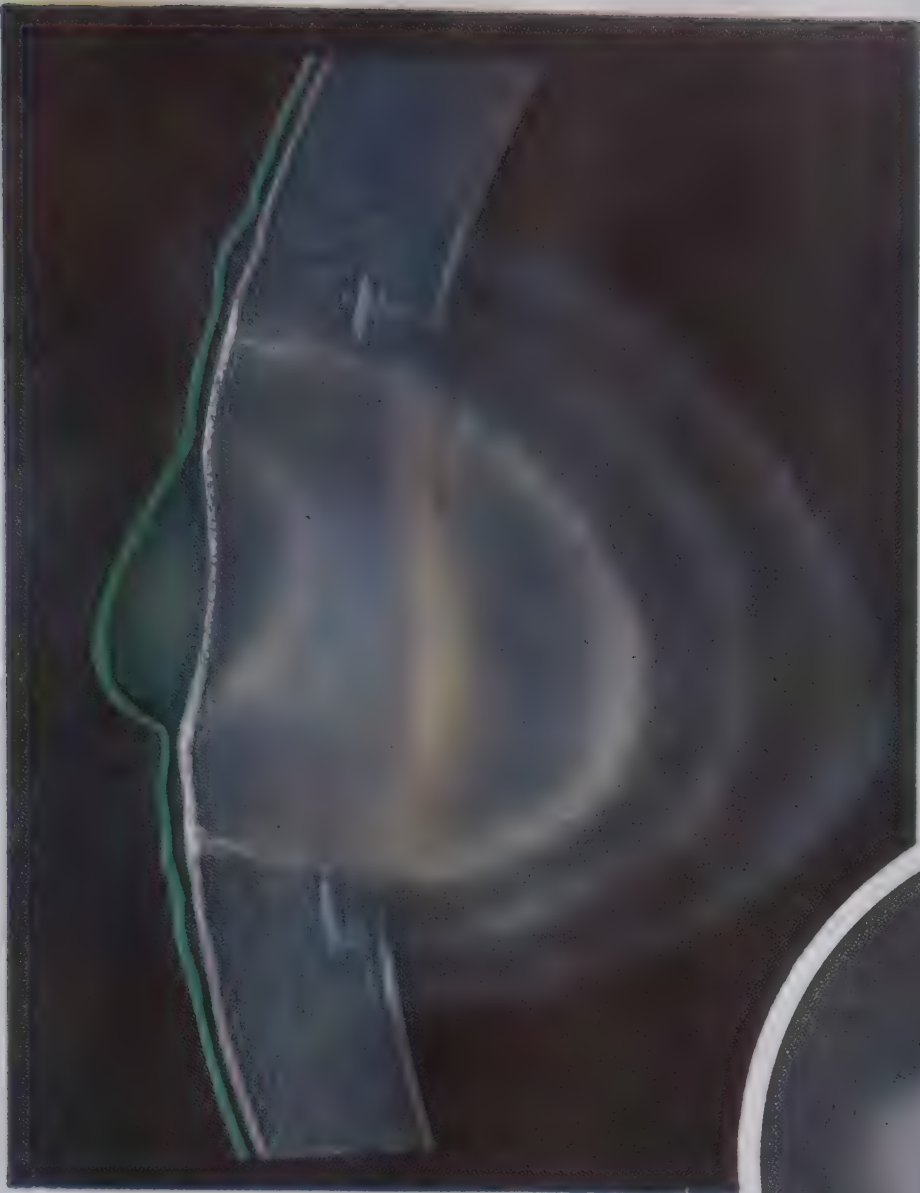
FIG. 2. Serpiginous ulcer — active stage by sclerotic scatter.

FIG. 3. Serpiginous ulcer — active stage. Optic section reveals central raised bullous-like epithelial blister and concentric rings of infiltration.

FIG. 4. Abrasions of the corneal epithelium by retro-illumination. (Fluorescein staining.)

FIG. 5. Marginal ulcer in a senile individual. Diffuse illumination.

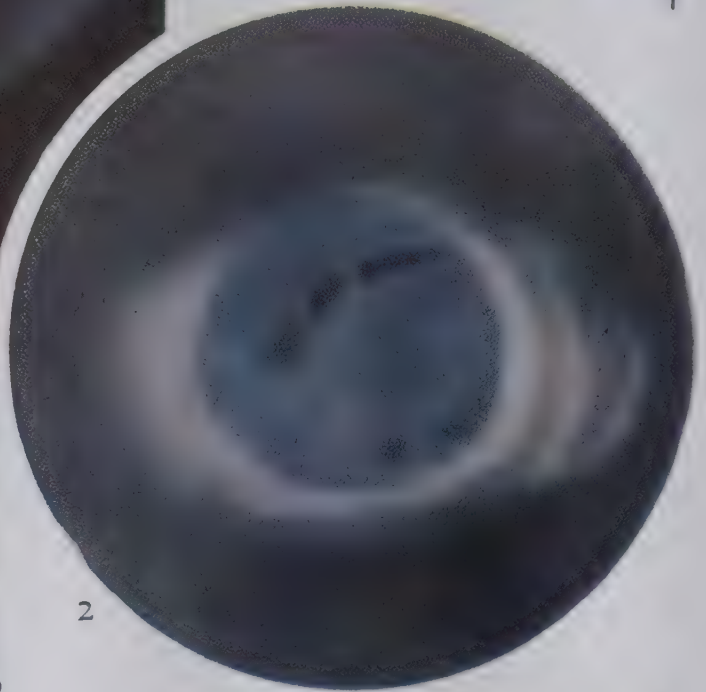
FIG. 6. A stained abrasion of corneal epithelium seen by sclerotic scatter.



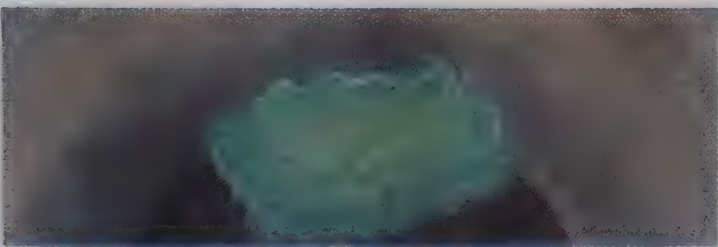
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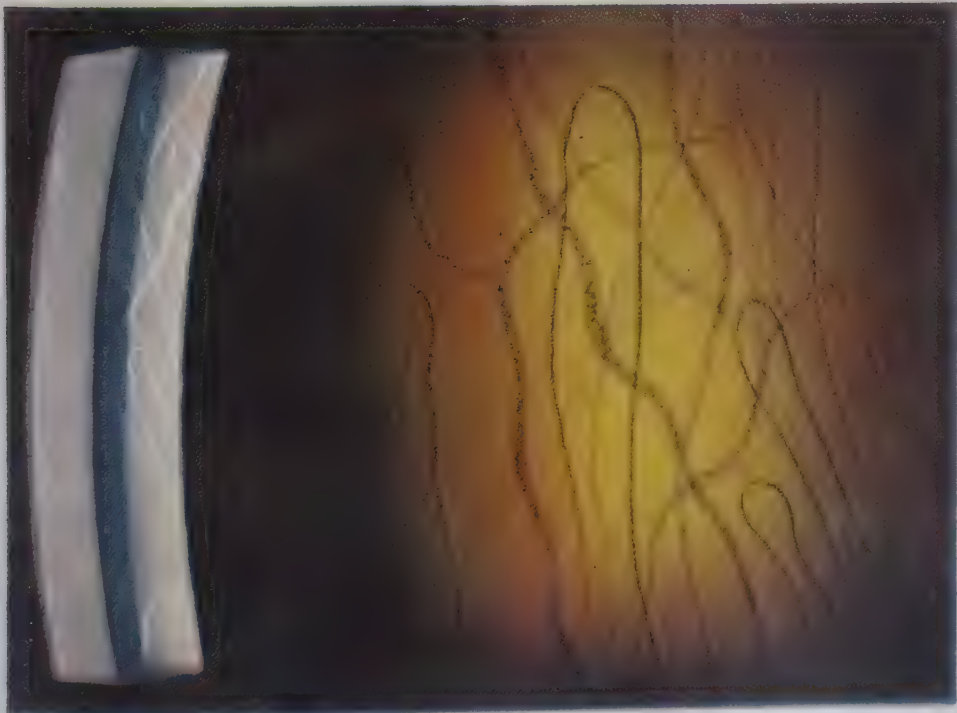
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formation of crescentic (catarrhal ulcers) or irregularly rounded opacities (acne rosacea).^{*} A breakdown of the devitalized overlying epithelium results in erosion and ulcer formation, which may or may not become secondarily infected. In *catarrhal ulcers*, the site of the ulcer may correspond to that occupied by an arcus senilis; similarly there may be a clear superficial area between it and the limbus.

In benign cases, there is usually a faint nebulous opacity, corresponding to the shape of the initial infiltration, which has little tendency to spread. Vascularization may be minimal but as a rule fine capillary loops extend toward the ulcer from the pericorneal vascular arcades. In the aged (Plate XXXVI, fig. 5) such a marginal ulcer may become indolent with minimal opacification but considerable loss of corneal substance. Indirect illumination or sclerotic scatter reveals a small narrow circular relucant ring with an almost clear central crater. This clear central area may be so depressed from loss of parenchymal substance and thinning that the anterior and posterior limits of the optic section seem to be in contact with one another.

Mooren's Ulcer (Ulcus Corneae Rodens, Rodent Ulcer, Chronic Serpiginous Ulcer). Mooren's ulcer is a rare type of idiopathic, painful, marginal ulceration, found in patients of advanced age. An outstanding feature is its slow but insidious progress over the entire corneal surface. It may occur in one eye or in both eyes, but not simultaneously. It is superficial, and perforation caused by primary lesions does not occur. However, perforation following secondary infection has been reported.

The lesion begins with a grayish infiltration below the epithelium (the site corresponding to that occupied by an arcus senilis) parallel to the limbus, or as a raised gelatinous infiltration contiguous to the sclera. The infiltration, which tends to progress slowly centrally, may partially or completely surround the cornea but it seldom penetrates deeper than the anterior third of its thickness. At the same

^{*} The more severe changes occurring in this type of ulcer have been described under keratitis of acne rosacea and phlyctenular keratitis.

time a marginal ulceration with the formation of a shallow peripheral sulcus develops.

This erosion undermines Bowman's membrane and forms a grayish crescentic thickened advancing border, which is loose and overhangs the floor of the ulcer. Immediately in front of this overhanging ledge, a narrow preceding relucet area is produced by epithelial edema and fine punctate infiltrates. The direction of progression is toward the center of the cornea, which remains clear until attacked by the advancing ulceration, but the advance may be greater in one sector than in others. At the same time, healing begins at the limbus and follows behind the advancing border. This leaves a rather dense scar, which is highly vascularized (Plate XXXIX, figs. 1, 2, 3).

The scar when viewed in optic section reveals a somewhat uneven precorneal film line and epithelium, beneath which there is a dense irregular cicatrix, involving Bowman's zone and the superficial parenchyma. When the optic section is directly centered over the ulcerating border, a sharp change in corneal thickness is observed. Marked thinning takes place at the floor of the ulcer (sulcus) and in the scarred and infiltrated area. The advancing cicatrization and vascularization do not stop the progression of the eroding border until the entire cornea is covered. Some cases have been described, in which the spread has been toward the sclera, causing formation of an elevated reddish perilimbal ring.

Fuchs¹⁰² reported a case, in which the inflamed eye showed a yellow ring, situated in the center of the cornea, somewhat wider and slightly elevated on the nasal side. The edge of the yellow ring was undermined; the center of the cornea was transparent, while the periphery exhibited a pannus-like vascularization.

Atheromatous Ulcer. The so-called "atheromatous" ulcers develop in old leukomas in which degenerative hyaline and calcareous changes have taken place. Owing to the lack of resistance and vitality of these tissues a small initial superficial ulcer may extend deeply and cause extensive necrosis with slough. Perforation with loss of the eye has been known to occur. If the original leukoma is not large and the surrounding normal cornea is resistant, the process

may be limited and only a small superficial sequestration may occur, followed by healing. Such localized processes may be followed by others in adjoining portions of the leukoma. Leukomas of long standing may undergo secondary ulceration at any time.

KERATOMYCOSIS

Infection of the cornea by the common fungi has been reported in diverse instances by many authors. It seems to be intimately related to superficial trauma of the cornea caused by foreign bodies, contaminated with spores of the fungi.

Unfortunately, accurate biomicroscopic descriptions of these lesions are few in number. The fungi most frequently implicated are *aspergillus fumigatus*, *streptothrix actinomyces*, *streptothrix nigricans* and *verticillium graphii*. The lesion caused by these agents is typical. It appears as a grayish necrotic torpid ulcer, usually central in site and surrounded by a fairly sharp and yellowish demarcation line. The floor of the ulcer is covered by the necrotic slough which may drop off leaving a fairly deep ulcer. The periphery of the ulcer (yellow line) appears gutter-like and represents marked leukocytic infiltration around the lesion.

Although perforation of the cornea and intra-ocular extension is rare, hypopyon may occur. In some instances, there is a milder form near the limbus, attended by a fascicular type of vascularization. Dense scar formation is the result of most cases of keratomycosis.

CORNEAL SCARS: CICATRICAL PROCESS OF THE CORNEA

Corneal scarring follows loss or destruction of parenchymal substance because in this tissue the process of healing is accomplished by replacement with opaque fibrous tissue. Only in the epithelium or endothelium can there be complete regeneration and hence no residual marks or scars. The term "opacity" is often applied somewhat loosely to all areas of increased relucency, irrespective of their nature, including those due to edema, infiltrations and folds, as well as scars. Although clinically when one refers to a corneal opacity

one implies a scarring process; strictly speaking, every scar is an opacity but every opacity is not a scar. Consequently, in biomicroscopy of the cornea it is best to avoid the term "opacity," which refers to an optical state rather than to an actual condition present, and to employ a specific pathologic designation.

Sclerotic scatter is the most effective form of illumination for disclosing the presence of corneal scars, especially small and thin scars. Even the faintest scars will cause dispersion of light and appear as a grayish opalescence. All scars stand out in direct focal illumination as highly relucet areas in the parallelepiped or the optic section.

Delicate faint scars (usually found in Bowman's zone) are referred to as "nebulae"; scars that are more opaque and localized are called "maculae," and those of intense, almost porcelain-white opaqueness are referred to as "leukomas." Adhesion or incarceration of iris tissue into a scar which has resulted from a perforating injury or ulcer is known as "adherent leukoma." Extensive scarring and thinning of the cornea may produce the bulging (from intra-ocular tension) known as "corneal ectasia." Ectasia with incarceration of the iris, either partial or complete, results in an anterior staphyloma, especially after a large amount of fibrous tissue replacement.

Epithelial regeneration always takes place in superficial ulcerative or traumatic lesions, however extensive they may be. Consequently, scars are covered with epithelium, except when recurrent or fresh epithelial erosions or ulcerations occur. Scarring results only when Bowman's membrane or the substantia propria is affected.

The importance of the narrow beam (optic section) for the correct interpretation of corneal scars is again emphasized because it affords the only means for their exact localization and for the determination of their thickness. Observed in the parallelepiped, a scar may appear to have a depressed or irregular surface. But inspection with the optic section may reveal the depression to be filled out by regenerated epithelium. Since the epithelium is non-relucet (optically empty) and nonrespersive, it cannot be seen in the parallelepiped (Fig. 241), while in optic section the precorneal film line, especially when it is stained with fluorescein, is seen to

conform with the general corneal curvature or to lie directly in front of a widened dark area, representing the thickened regenerated epithelium.

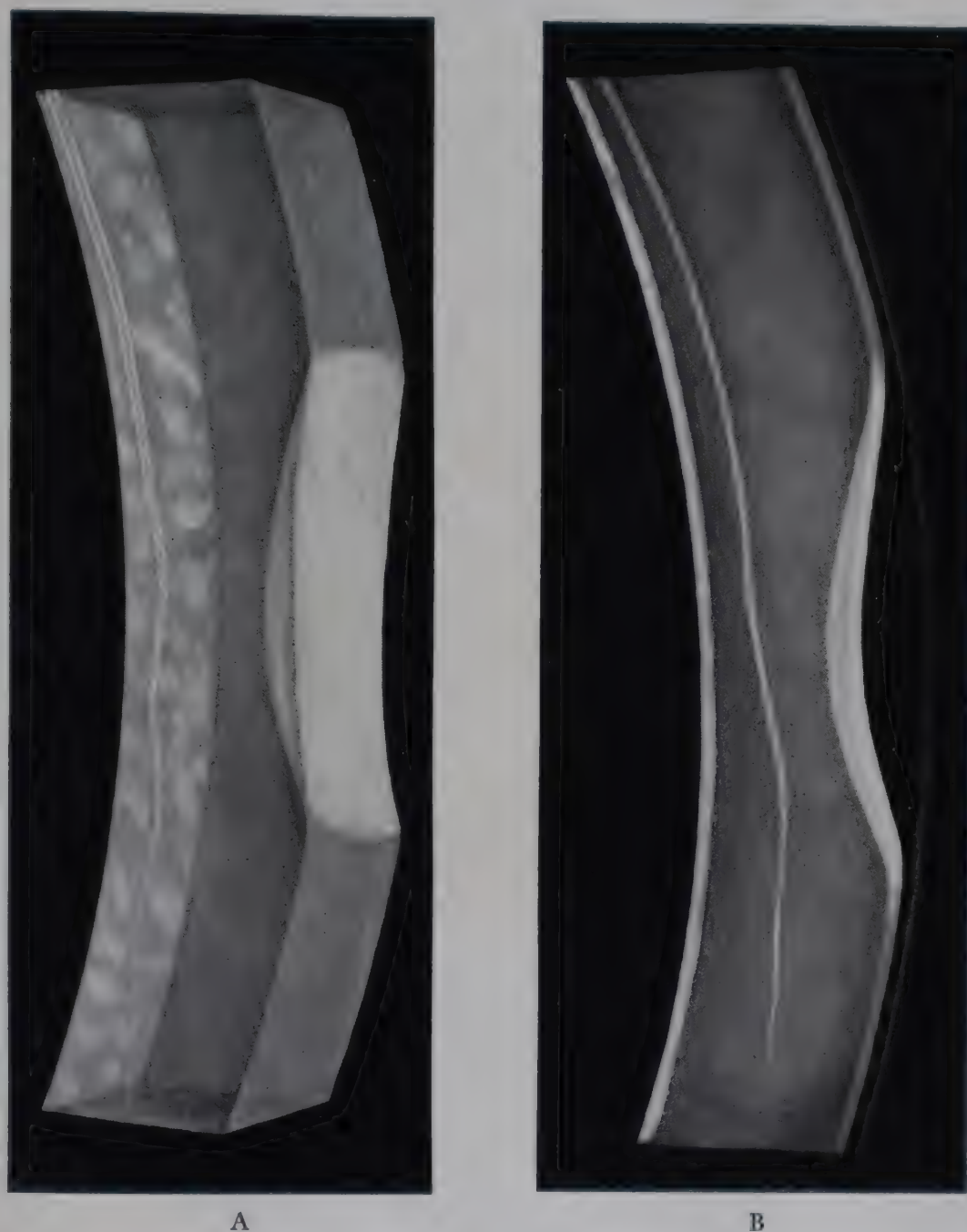


FIG. 241. A. Corneal scar following ulcer by direct focal illumination. B. The same lesion in optic section. Fluorescein staining of film line reveals the filling in of the defect in Bowman's zone with epithelium.

The corneal thickness is always thinned in the region of a scar. In other words, the reparative processes (scarring) never restore the cornea to its original thickness, but a process of filling by epithelial overgrowth tends to re-establish the surface curvature.

From a diagnostic standpoint, the shape of a scar may be etiologically significant (e.g., herpetic scars). Scars may vary greatly from

small irregular or annular opacities to large diffuse irregular or discoid forms, extending over the entire cornea, involving some or all of its layers. Certain diseases produce scars with definite char-

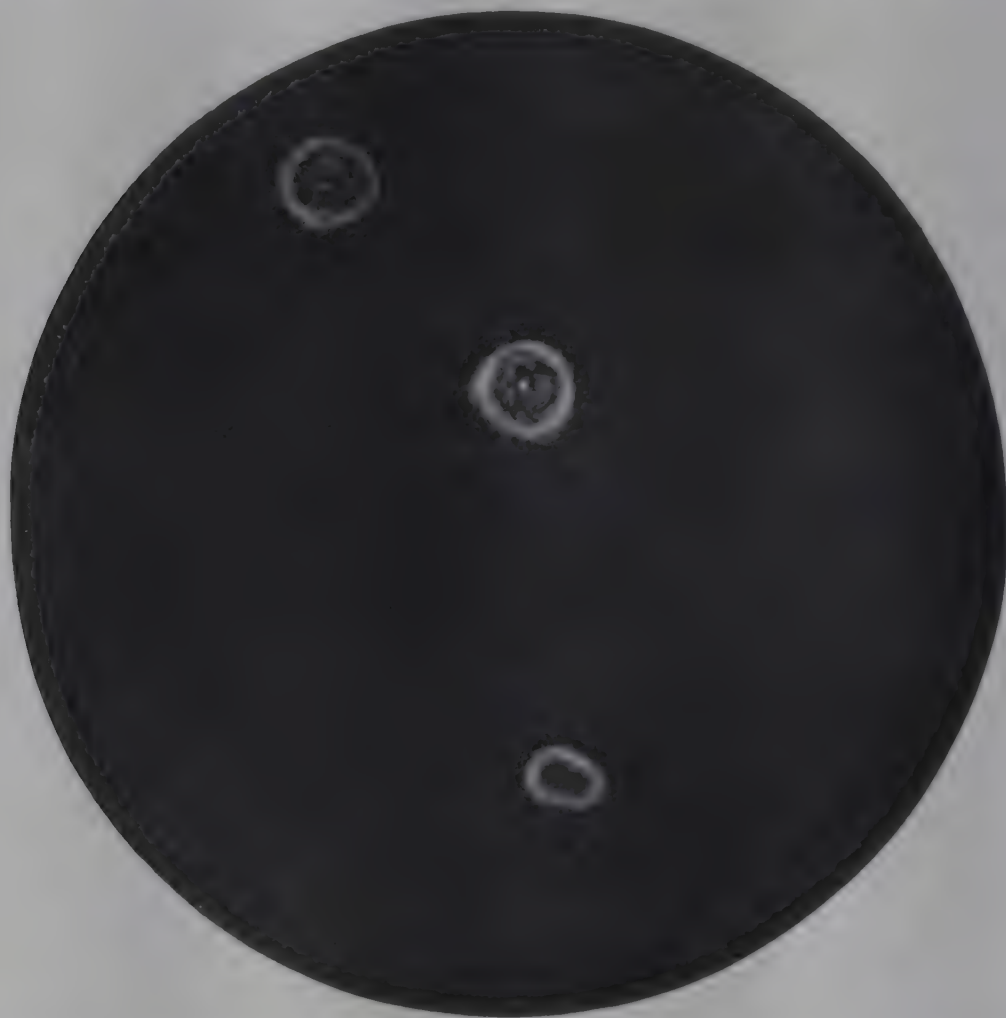


FIG. 242. Maculae of the cornea. Small annular scars in Bowman's membrane following extraction of foreign bodies embedded in the cornea of a stone worker (sclerotic scatter).

acteristics. An example of this is the superficial annular scar following foreign bodies embedded in Bowman's membrane (Fig. 242). These are characterized by a dotlike central white area surrounded by an annular nebulous haze. Following herpes cornea, groups of small, superficial, irregularly annular opacities with central areas of increased density may be seen (Figs. 243, 244). Band-shaped keratitis also forms typical superficial fenestrated scars (page 343). Incised or perforating wounds from any cause leave scars extending through the entire corneal thickness. These may be uniformly linear or irregular. Hess^{150, 151} described a peculiar hyaline filament formation, in discission wounds. These resembled slightly the filaments seen in filamentary keratitis but were acellular. Hess believed that they were formed by the minute escape of vitreous from the wound.



FIG. 243. Discrete superficial scars following herpes of the cornea by sclerotic scatter.



FIG. 244. Optic section through a healed herpetic scar with central calcareous deposit.

Vascularization varies greatly. In many instances, no vessels may be present. In others, the scar may be highly vascularized. In superficial lesions the vessels (if present) tend to lie in the superficial



FIG. 245. Clearing stripes in corneal scars. In some cases these clearing stripes form around the vessels.

planes of the cornea. In more extensive lesions, owing to parenchymal destruction, both deep and superficial interanastomosing vessels may be present. Although the vessels may penetrate a scar, as a rule they tend to arborize around it. In old scars the vessels may become obliterated, appearing as opaque whitish lines by direct focal illumination, and as yellowish tubes by retro-illumination.

Although corneal scars are permanent, they tend to change with time. In the young, a rather thick grayish opacity with indistinct edges may later thin out, becoming less opaque, bluish white in color, more sharply defined and less vascularized. This is a conse-

quence of the tendency of connective tissue in the cornea to assume gradually the appearance and characteristics of the normal corneal parenchyma. As a matter of fact, it is for this reason that in many instances, it is difficult to find histologic evidence of a corneal scar which has been conspicuous in life.

Fuchs¹⁰⁴ noted in the clearing of a scar the appearance of dark bands traversing the scar in geometric patterns (Fig. 245). At first, he attributed these clearing stripes to lymphatic absorption and then¹⁰⁵ to the formation of new, clear corneal tissue. However, later workers have shown that these clearing areas follow the course of blood vessels. Frequently, small obliterated vessels are seen as tracts in dark clearing stripes. By direct focal and diffuse illumination they appear as clear, dark linear spaces, surrounded by relucant tissue. These tracts may be parallel, intersecting, angulated or radial. Clearing stripes are usually found in large scars.

As previously mentioned, in most instances corneal scars are covered by intact epithelium (Fig. 246 A and B). However, in certain superficial scars, subepithelial changes may be observed, such as thickening in the form of whitish gray bands. This results in surface deformation. The raised epithelium over such a lesion may become finely abraded, causing irritation. These changes may be seen in superficial punctate keratitis, herpetic keratitis, keratoconjunctivitis eczematosa, and rosacea keratitis. In other instances, cystic degeneration of the scar surface may follow exacerbations or faulty healing. Small, hardly visible grayish dehiscences may be seen in Bowman's zone. The overlying epithelium may be edematous and lead to the formation of vesicles and bullae. Epithelial erosions and ulcerations from their rupture may ensue.

In certain superficial affections of the cornea (keratitis neuro-paralytica and lattice-form keratitis), a grill-like striate opacity is apparent, which has been attributed either to involvement of the corneal nerves or to tears in Bowman's membrane. On the surface of many superficial scars, particularly in the interpalpebral zone of the cornea, an overlying, subepithelial irregular pigmented line is found (Stähli's line) (page 381). Such pigmented lines can be seen

even in avascular scars. In old staphylomatous corneas, melanophores from the uveal tract frequently migrate into the scar.

Large scars generally exhibit some form of late degenerative

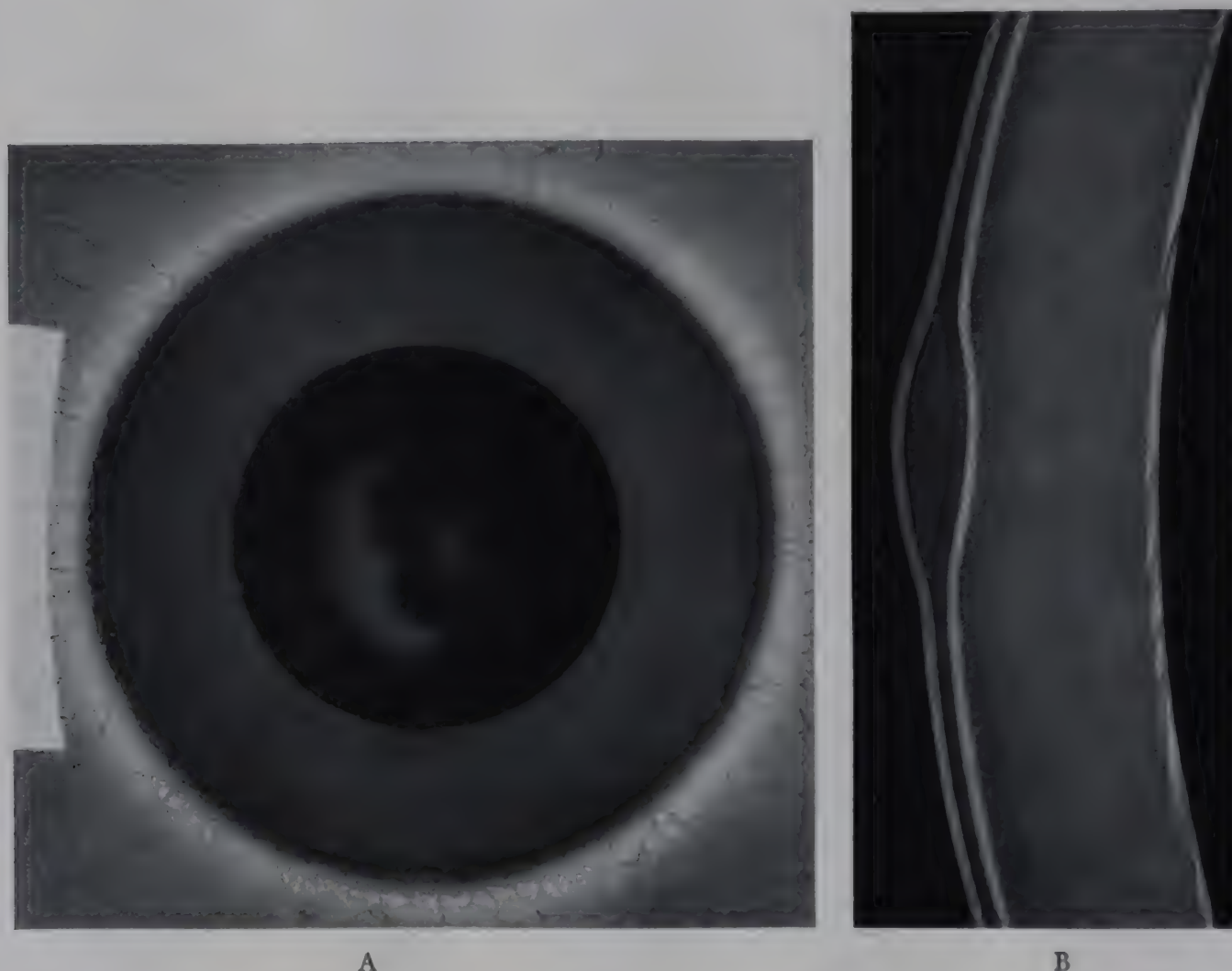


FIG. 246. A. Superficial corneal scars by sclerotic scatter. B. Optic section through the larger scar showing raised and thickened epithelium, relucant Bowman's zone and folds in Descemet's membrane.

change. Calcareous depositions are the most common but hyaline and fatty or lipin changes are also seen. Extensive hyaline or calcareous degeneration may lead to the formation of plaques, which at times may become spontaneously detached (Fig. 247). The calcareous deposits appear as dead-white granules or plates, while hyaline material has a yellow color. In the fatty and lipin degenerations, definite needle-like crystalline forms may be seen in the yellowish matrix. Likewise, varicolored crystals, supposedly of cholesterol, may be associated with dense white corneal leukomas. Crystals of uric acid or urates and hematin have also been described. Diverse crystals, some white, others iridescent, varying in contour (flat,

rhomboidal, or needle-shaped), the chemistry of which is unknown, are seen in many corneal opacities. Rarely, a sequestering process attends necrosis of the superficial portions of the scar with consequent

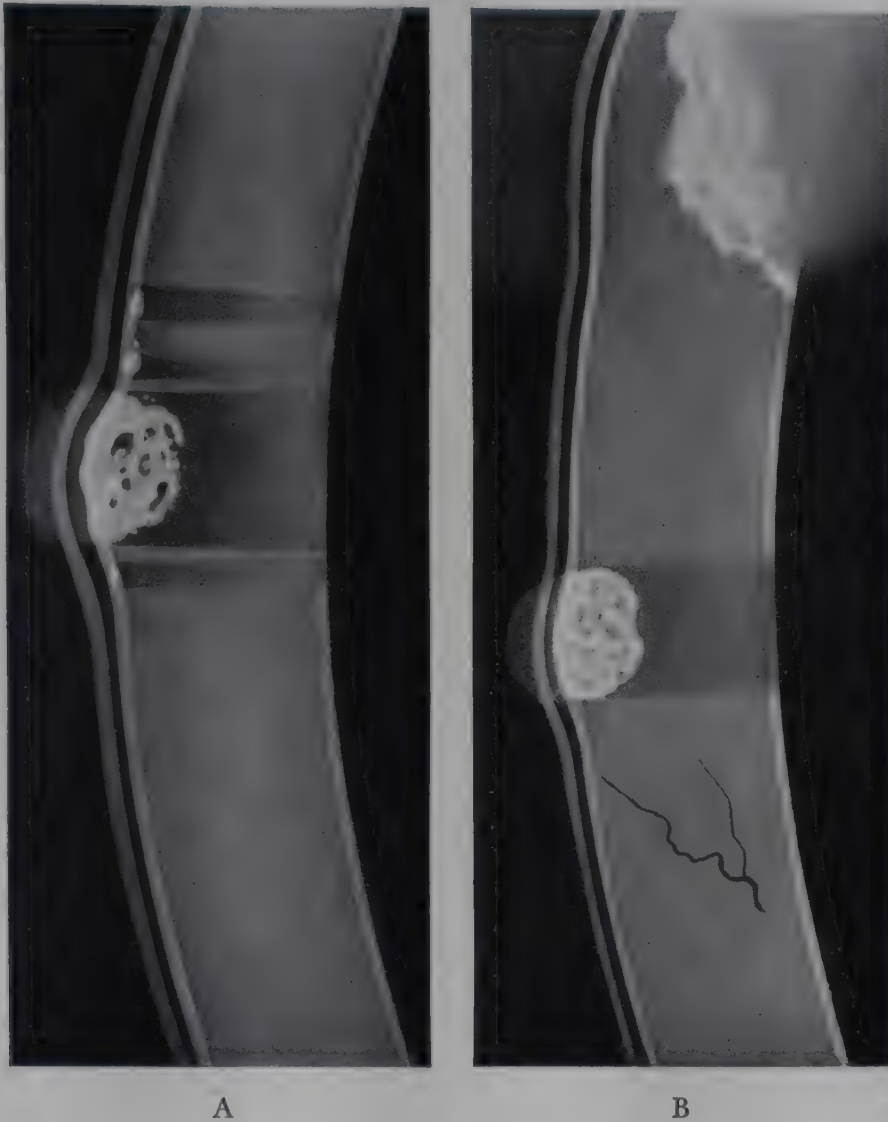


FIG. 247. Optic sections. A. Superficial scar with calcareous deposits. B. Similar type of scar near limbus associated with deeper lesion.

ulceration (atheromatous ulcer). Proliferative changes are occasionally present in dense scars. Fibroma or keloid development has been noted. A case of the latter was seen in a young negress after recovery from an acquired gonorrheal ophthalmia with complications. A large progressive keloidal scar developed in the opaque cornea. This patient had cutaneous keloids also.

In interstitial keratitis (page 506), the disease process in the deeper layers results in infiltration and parenchymal destruction, leading to a more or less diffuse, hazy, soft type of scar, to which clearing stripes may give a digitiform appearance. In other instances,

long dense linear streaklike opacities are found. Irrespective of the changes in the middle layers of the parenchyma, a permanent reluctant Descemet's zone persists in this condition (Fig. 227). Except with the biomicroscope, this reluctant layer may appear thin, and be difficult to detect.

A corneal opacity, shaped like a mushroom cap, is formed at the limbus in front of a progressing pterygium, dermoid, nevus, or following or adjacent to an area of scleritis. After the removal of a pterygium, dermoid or nevus at the limbus, a thin superficial corneal scar results, which may be pervaded by sinuous capillaries and may be associated with facetting of the surface.

Chapter Fifteen

TRAUMATIC INJURIES TO THE CORNEA

BECAUSE of its exposed position, the cornea is especially vulnerable to injury. Extensive injuries to the cornea are usually associated with varying degrees of damage to the deeper intra-ocular structures. The frequency with which lesions are overlooked by ordinary methods of examination makes biomicroscopic examination of traumatized eyes imperative. As pointed out by Davidson,⁵⁶ "the immediate or retrospective diagnosis of a recent eye perforation or rupture presents little difficulty, and the medico-legal problems arising in connection with them, compared with those in contusions, are correspondingly easier to solve. In spite of this, however, there are encountered in the examination of eyes for workmen's compensation, too many overlooked intra-ocular foreign bodies; many eye perforations and ruptures, too, are undiagnosed, often having been regarded as merely corneal abrasions or opacities or conjunctival lacerations. It is quite true that conjunctival hemorrhages and lacerations may easily hide a limbic or scleral perforation, with or without the retention of a foreign body. Difficulties present themselves in the retrospective diagnosis of older perforations. Very old corneal perforations are not always recognizable as such. Biomicroscopic demonstration of conjunctivo-scleral adhesions, which prevent the conjunctiva from gliding freely over the sclera, of subconjunctival foreign bodies, of conjunctival scars and linear pigmented scleral ruptures, is often very helpful."

CONTUSIONS

Depending on the violence of trauma, contusions may produce corneal lesions varying from epithelial and parenchymal edema to

actual ruptures of the various layers of the cornea. After a mild contusion the only change noted may be transient edema of the epithelium, which may be associated with small whitish relucant dots. In other instances, sclerotic scatter reveals a transient discoid haze, which is seen in optic section deeply situated and accompanied by folds in Descemet's membrane. This haze usually does not obscure the view of the deeper portions of the eye.

Ruptures of Descemet's membrane permit imbibition of aqueous fluid by the corneal parenchyma, and cloudy edema of the cornea results. The torn ends of Descemet's membrane may become rolled up and present a characteristic scroll-like appearance (page 422). Ruptures of Bowman's membrane result in linear or star-shaped opaque figures. Total, perforating ruptures of the entire corneal thickness may follow violent contusions, with the attendant complications usual to such accidents.

Diffusion of blood between the corneal lamellae in hyphema causes an irregular reddish zone. Disintegration of the red cells produces a gray or chocolate-brown color, which later may become greenish in hue. At times crystalline elements may be present (Plate XXIII, fig. 6).

Ultimate resolution and clearing follows most mild contusions of the cornea. Severe contusions may be associated with deposits of pigment on the posterior corneal surface (traumatic iritis). These may be diffuse or annular. In the latter case, when the pigment lies directly in front of the pupillary area it forms a ring, corresponding to Vossius' ring on the anterior lens capsule (traumatic annular episcapsular pigment deposit). Occasionally, after prolonged tight bandaging, a mild transient epithelial edema may occur in association with ocular hypotony.

ABRASIONS

Abrasions of the corneal surface follow severe contusions. They are also seen following traumas that appeared trivial at the time of injury (Plate XXXVI, figs. 4, 6). A scratch by the sharp edge of a

sheet of paper or by the fingernail of a baby, the sudden swish of foliage against the eye, the prick of a thorn, foreign bodies or calcareous deposits embedded on the tarsus of the upper eyelid, and the irritation caused by displaced cilia are examples of various types of traumas which may cause epithelial abrasions. Likewise, failure to keep the eyelids closed after cocaineization may cause a similar lesion of the desiccated epithelium. Abrasions may appear immediately after injury or their appearance may be delayed. Once an abrasion has occurred, repeated, apparently spontaneous, recurrences may ensue; for example, a painful recurrent erosion may develop suddenly upon opening the eyes in the morning.

Biomicroscopic examination of corneal abrasions and erosions is important, not only for diagnostic purposes but also because of the medicolegal aspect these cases may assume. Because of extreme pain and photophobia it is usually necessary to anesthetize the cornea in order to facilitate biomicroscopic examination. Staining with a 2 per cent solution of fluorescein will reveal the lesion. A greenish stain results, corresponding to the size of the denuded area.

An unusual fluorescein staining reaction may be seen in certain erosions. Since the dye seeps under the loosened epithelium bordering an erosion, a subepithelial staining occurs, which may give the impression that the stainable erosion is larger than it actually is. This seepage of the dye produces a double line of fluorescein coloration in the optic section immediately adjoining the erosion. The anterior line is the stained precorneal film line, while the underlying one is caused by the seepage of the dye under the epithelium.

When unstained, the appearance of an abrasion is characterized by a defect in the epithelium, the edges of which are slightly hazy. Rarely, the floor of a large erosion may have a grayish appearance owing to the changes in Bowman's zone. In optic section an interruption of the precorneal film line is seen over the affected area. Irregular brilliant reflexes caused by irregular reflection are noted. Healing of most abrasions is rapid unless they are infected. In from twenty-four to forty-eight hours macroscopic evidence of an abra-

PLATE XXXVII

FIG. 1. Corneal scars following incised wound. Below, the scar extends through the entire corneal thickness.

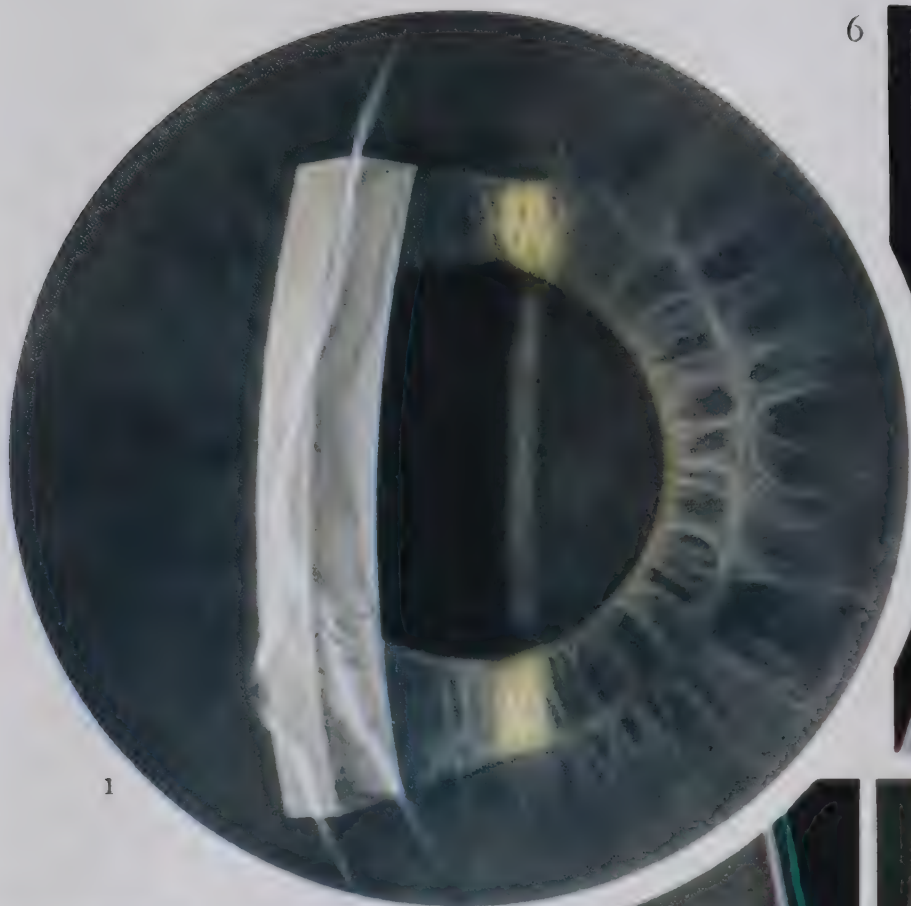
FIG. 2. Fresh lacerations of the cornea caused by thumb nail. Appearance of lacerations in parallelepiped. Superficial and deep penetration of fluorescein staining.

FIG. 3. Endothelial surface (specular reflection) in above case (Fig. 2) showing partial staining of endothelial cells (below) with fluorescein and folds in Descemet's membrane.

FIG. 4. View of one of the lacerations (Fig. 2) by retro-illumination, high magnification. Edges are relucant.

FIG. 5. A tiny particle of coal in Bowman's zone as seen by optic section causing surface irregularity. Note shadow cast by foreign body throughout the thickness of the optic section.

FIG. 6. Optic section showing numerous deposits of coal dust below the epithelium.



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sion may disappear. However, more detailed examination with the biomicroscope may reveal small faintly relucant dots over the site of the abrasion for some days. Increased visibility of the corneal nerves also may be noted.

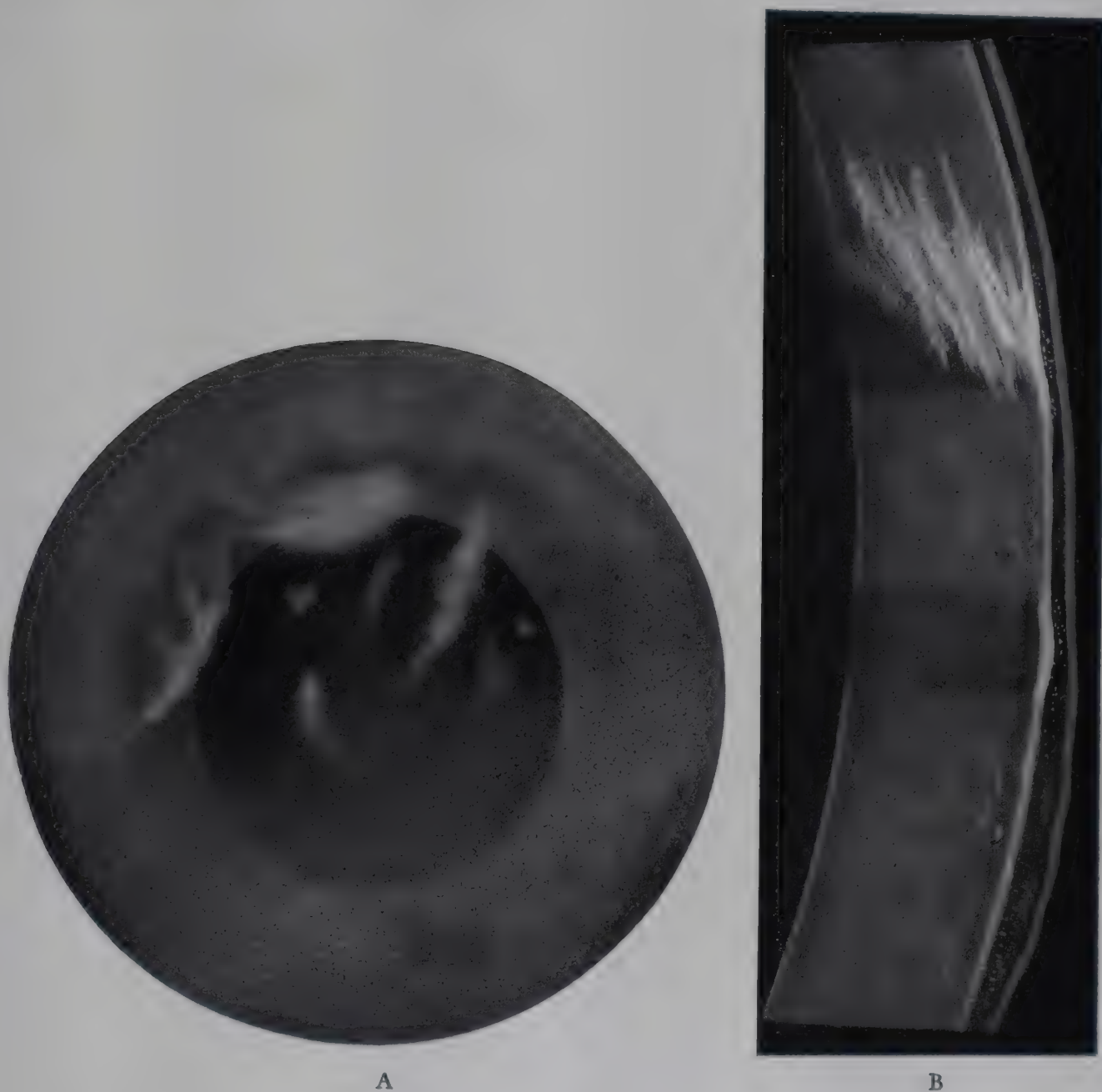


FIG. 248. Perforating wounds of the cornea. A. By diffuse illumination. B. Optic section through one of scars in A showing ragged opacity extending through corneal thickness.

WOUNDS

The nature of corneal wounds depends on the violence of the blow and the nature of the instrument causing the injury (Fig. 248). Obviously, since no two injuries are alike, the resulting changes do not conform to any strict formula. Most perforating corneal injuries are caused by sharp objects or small penetrating foreign bodies,

and therefore the corneal scars tend to appear as linear opacities. Consequently, the presence of linear scars which pass through the layers of the cornea suggests an antecedent perforating trauma. Corneal wounds may be nonpenetrating or penetrating. In penetrating wounds all the corneal layers are traversed by the damaging object (Plate XXXVII, fig. 1). In nonpenetrating wounds there may be a solution of continuity of any or all of the layers of the cornea up to Descemet's membrane. The chief point of differentiation is that in nonpenetrating wounds the opening does not extend into the anterior chamber. Freshly incised wounds may be represented only by a line of tissue discontinuity. If examined shortly after the injury, it is seen that because of healing processes (e.g., edema) the edge of the wound is relucant (Plate XXXVII, fig. 4). Staining with fluorescein at this stage may reveal an irregular film line and penetration of the dye as far as the endothelium (Plate XXXVII, figs. 2, 3). Inclusion of foreign bodies or debris or epithelial downgrowths between the lips of the wound may interfere with the process of surface epithelialization and leave an irregular surface (Plate XXXVII, fig. 5). Such faulty healing may cause a cystoid scar (cystoid cicatrix).

A sharp pointed instrument or a perforating foreign body may cause a scar which has a straight track similar to that of an incised wound. In injuries caused by a needle or thorn the track tapers in its deep portions. Such scars, when examined with the biomicroscope, often appear granular.

It must be remembered that only the epithelium and the endothelium regenerate and that any wound involving Bowman's membrane and the corneal parenchyma heals by cicatrization, and no matter how minute, biomicroscopic evidence of its presence remains. Sclerotic scatter is a useful method of revealing faint scars resulting from such injuries.

Frequently, foreign body detritus may be incorporated in the scar. Irregular scars may result from nonpenetrating ragged lacerations. A superficial laceration of the cornea may result in a Y-shaped or crucial type of scar (Fig. 249). In the early stages there is considerable edema in the neighboring epithelium and parenchyma.

The edges of the wound are bordered by opalescent areas. After cicatrization, the optic section always shows variations in corneal thickness in the region of the scar while extensive thinning of the



FIG. 249. Superficial laceration of the cornea, Y-shaped scar.

cornea and ectasia of varying degrees may result. Such wounds heal rapidly unless secondary infection occurs; in this case there is an increasing grayish infiltration of the borders of the wound. Severe secondary infection may terminate in hypopyon ulcer with its attending complications. In these cases, vascularization of the cornea is likely to occur during the healing stage.

Although folds of Bowman's membrane may occur with non-perforating wounds, they are not as common as folds in Descemet's membrane.

Perforating wounds of the cornea may result if the entire thickness is traversed by a minute metallic body, traveling with high velocity. Flying fragments of glass likewise may pass through the cornea. Opacification of the walls of the track reveals the path taken by the perforating object. As in a nonperforating injury, the size and shape of the resulting lesion depends on the force of the blow and the nature of the penetrating instrument or foreign body. Perforating wounds are usually accompanied by folding of the glass membranes. Koby pointed out that alterations in the zone of specular reflection on the posterior surface indicate that perforation has probably been complete. In all tears of Descemet's membrane, whatever the cause, a tendency to backward rolling of the torn ends is demonstrated by the loose edges in the anterior chamber. In other cases, the membrane may not become detached but a posterior opacity may develop. The presence of intra-ocular foreign bodies should be considered whenever small linear scars are found extending through all layers of the cornea. A hole in the iris or a lenticular opacity, in line with such a scar, usually indicates the path of a penetrating foreign body. In all cases of linear scars extending through the corneal thickness it is advisable to make roentgenographic studies to rule out the presence of radio-opaque foreign bodies.

In large perforations with extensive laceration of the cornea, the lips of the wound may override. This leads to the formation of irregular scars. With small perforations (e.g., needle punctures) rapid closure of the wound may result in only a minimal amount of parenchymal imbibition of aqueous fluid, whereas large lacerations are accompanied by severe corneal hydrops. In the latter case, after cicatrization, optic section reveals varying amounts of distortion and thinning.

A variety of complications follow perforating injuries, depending on their severity and location. Prolapse or extrusion of any or all of the internal structures may lead to dire complications and loss of the eye. Secondary infections progress to corneal abscess and even to panophthalmitis.

Surgical incisions in the cornea are always accompanied by scar formation, which can readily be seen with the biomicroscope (Fig. 250). The type of scar depends on the instrument used



FIG. 250. Optic section through a corneal scar at the limbus following corneal section with Graefe knife for cataract extraction.

and the mode and the extent of the incision. The passage of a small needle-knife through the cornea leaves a delicate opaque track which tends to become slightly granular. However, following the needling of a secondary membrane, it is not unusual to see tags of vitreous adherent to the scar. With such tiny wounds, reaction is limited to minimal epithelial and parenchymal edema. Folding of the glass membranes rarely occurs. The amount of scarring following a keratome incision into the cornea depends on the obliquity of the cut. With a shallow anterior chamber the necessity of avoiding the iris frequently produces a markedly oblique shelving incision, which leads to lamellar splitting of the cornea and even separation of Descemet's membrane (Fig. 203). Such damage to Descemet's membrane may cause a spade-shaped opacity. Likewise, instruments inserted in the anterior chamber (e.g., spoon, spatula, forceps) may cause abrasions and permanent opacities in Descemet's zone. In corneal

section for cataract extraction with the Graefe knife the incision may be somewhat irregular because of an early collapse of the anterior chamber and consequent distortion of the cornea. This may

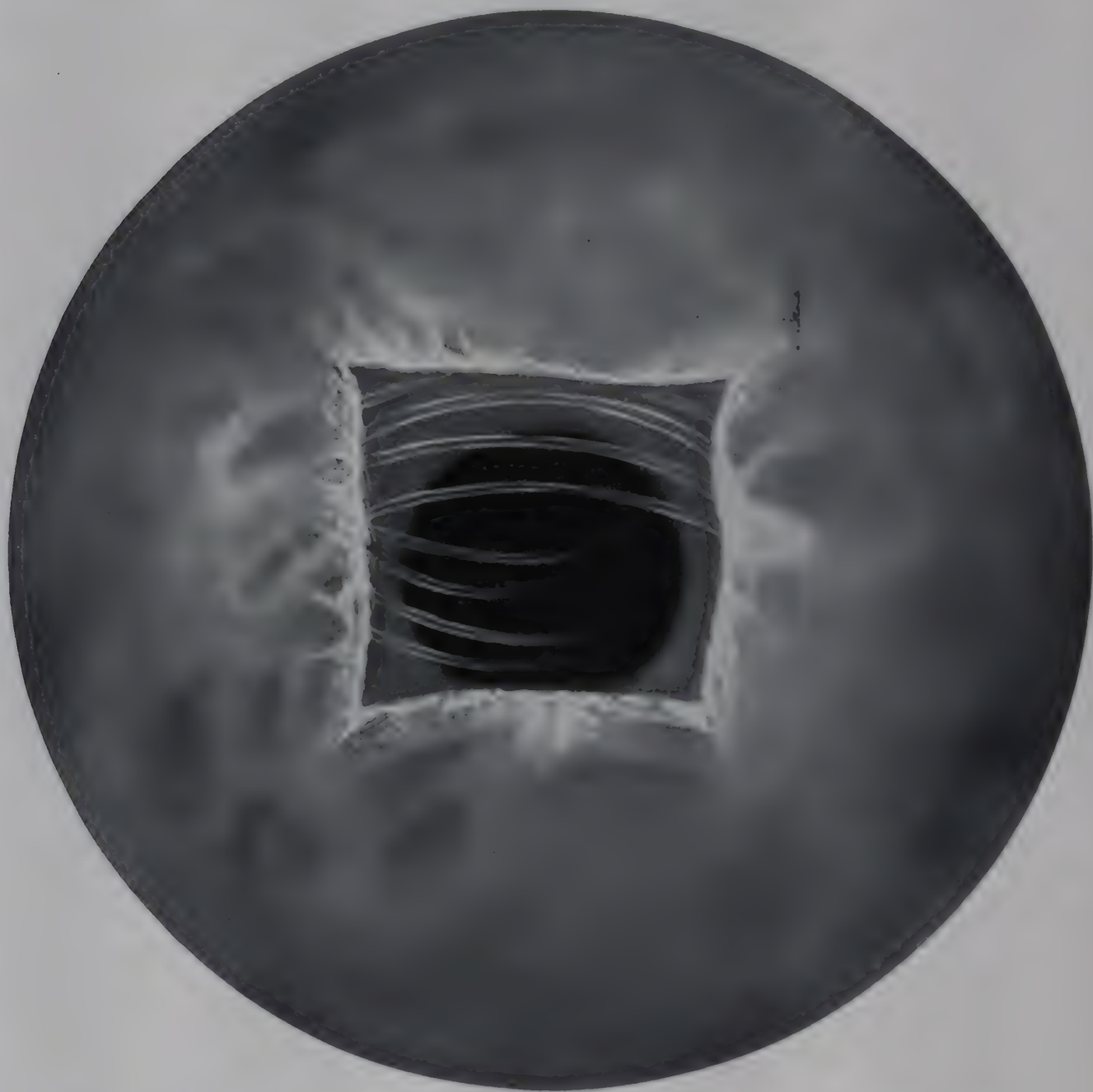


FIG. 251. Successful keratoplasty in case of diffuse corneal leukoma. Horizontal folds in Descemet's membrane seen in clear transplant. Vision, 20/30.

cause a variety of corneal scars, when the point of exit of the blade is in clear cornea. In one case in which the point of exit was in clear cornea, a clear vertical streak was seen in Bowman's zone surrounded by some delicate parenchymal opacities (Fig. 200).

One of the immediate sequelae of surgical procedures for cataract is extensive folding of Descemet's membrane, which, when marked, is called striate keratitis.

In total penetrating keratoplasty, whether the inserted graft be circular or rectangular the edges will be permanently outlined by a scar running through the corneal thickness. The surface of the graft is generally higher than the surrounding cornea (Fig. 251).

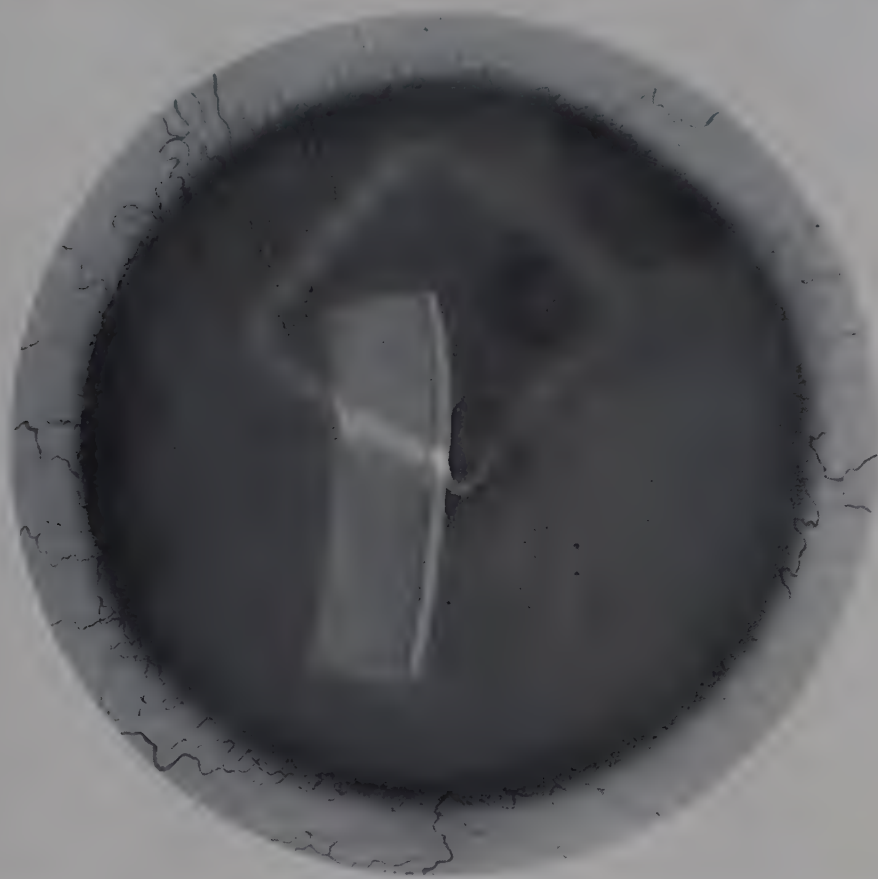


FIG. 252. Appearance of penetrating keratoplasty in direct focal illumination showing marginal scar through the corneal thickness. The square corneal transplant became opaque.

FOREIGN BODIES IN THE CORNEA

Undoubtedly, the most common ocular injuries are caused by foreign bodies, such as small particles of coal, dust or metal, becoming embedded in the cornea. Foreign bodies may be attached loosely to the epithelium or may be firmly embedded at any depth in the corneal parenchyma. The importance of biomicroscopic examination in such cases cannot be overemphasized. Examination with the optic section not only permits exact localization of the foreign body, but in most cases identifies the nature of the foreign substance. Moreover, I have found that corneal foreign bodies can be removed expertly under direct biomicroscopic view. An opaque foreign body throws a shadow posteriorly through the entire thickness of the cornea. This factor is useful in localization (Plate XXXVII, figs.

5, 6). It is rare for an ordinary small foreign body to penetrate deeply into the cornea. The great majority are found in the epithelium or slightly deeper in Bowman's zone. When the foreign body penetrates beyond Bowman's membrane, and the epithelium regenerates rapidly without infection, it may be tolerated in the cornea with little or no reaction; for instance, stone-cutters tolerate innumerable embedded particles of stone. Usually, there is a tendency for small sloughs to form around a retained foreign body; in this case considerable localized edema may be present. Edema may excite a reaction in the deep parenchyma with consequent folding of Descemet's membrane. Foreign bodies, which have penetrated Bowman's membrane, always leave scars. If the foreign body is promptly removed less scarring follows than if sloughing and ulceration occur. The typical resultant scar is usually seen in Bowman's zone as a small annular macula. Such subepithelial ringlike scars have a more relucet center in the form of a whitish spot and a less relucet halo (see Fig. 202).

Metallic foreign bodies, such as iron chips, are frequently surrounded by a reddish brown rust ring, which is usually seen on the day following the removal of the foreign body. If this material is not removed, it tends to slough out and a small ulcer forms.

Stone or metal workers frequently present the startling picture of a cornea studded with foreign bodies, which seem to be well tolerated. In these cases, as already mentioned, the foreign bodies are in Bowman's zone or deeper, and are covered by intact epithelium. Each foreign body may be surrounded by a narrow relucet zone. McDannald reported a case of well-tolerated gold particles embedded in the cornea following an explosion. They were seen as brilliant yellow golden particles in the anterior parenchyma. There was little reaction surrounding these particles. Similarly, coal fragments (Plate XXXVII, figs. 5, 6) or gunpowder grains have become embedded in the corneae following explosions. After the initial stage of irritation, these become well tolerated and have no inflammatory sequelae. Under magnification, coal particles are facettet and glistening.

A particle of glass embedded in the cornea may be identified easily with the biomicroscope. The refractile particle usually displays brilliant reflexes in direct focal illumination and although transparent will cast posterior shadows in optic section.

THERMAL, CHEMICAL, AND IRRADIATIVE INJURIES

Because of the difficulty inherent in examining recent injuries of this type, the value of biomicroscopy lies largely in the study of the end result.

Thermal injuries or burns of the cornea result from contact with flames, molten metals, scalding fluids, or freezing agents (e.g., exposure to cold, carbon dioxide, snow, liquid air). These injuries are usually associated with burns of the eyelids and conjunctiva. Even in slight burns, injection and chemosis of the conjunctiva usually occur. The damaging effects of such agents in the cornea depend on which of the corneal structures are injured. If the burn is superficial, only the corneal epithelium may be involved. The immediate effect is haziness and separation of the epithelium, which may become desquamated, resulting in a condition similar to erosion or abrasion of the cornea. In some instances there is little or no damaging sequelae; in others, vesicle formation may occur.

When the burn affects Bowman's membrane or the deeper stroma, a granular opacity results. In severe burns involving all the corneal layers, a porcelain-like appearance may develop and extensive necrosis of the cornea ensue. If the entire cornea is not lost, extensive scarring forms a dense leukoma.

Chemical burns of the cornea caused by acids or alkalies frequently occur in domestic and industrial life. As a rule, the acids cause immediate destruction of corneal tissue, whereas in the case of alkalies, particularly lye and ammonia, there may be a delayed reaction. Depending on the amount and concentration of the chemical, varying degrees of damage occur. In a case of a mild lye burn seen recently all conjunctival reaction had disappeared within two weeks but the cornea still revealed small punctate opacities in Bowman's zone over its entire area (Fig. 253). Usually, however, there

is an immediate and intense clouding of the entire cornea, which may increase until the cornea becomes white and porcelain-like. In severe cases perforation of the cornea with all the attendant

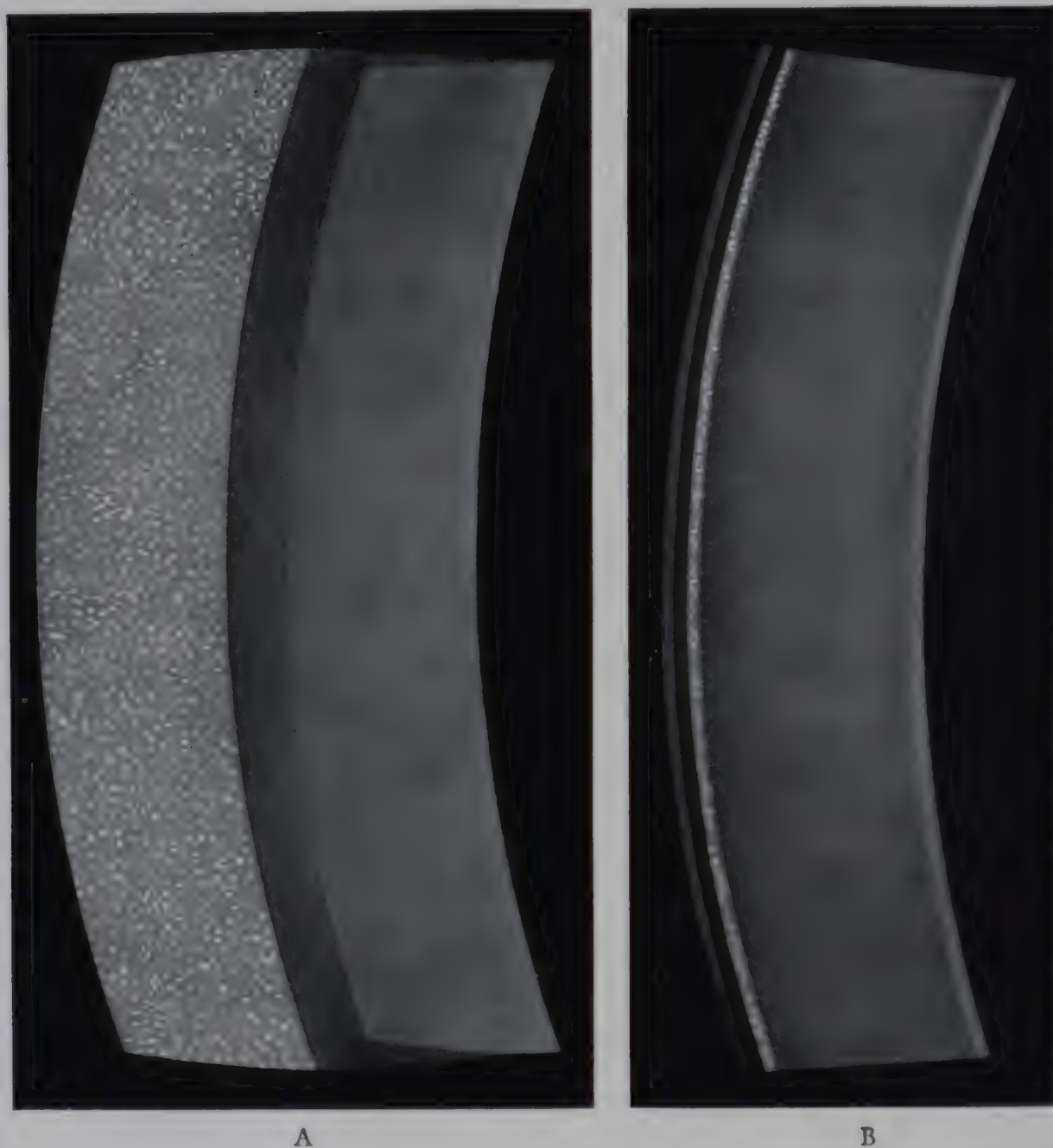


FIG. 253. Mild lye burn. A. Parallelepiped. B. Optic section. Superficial punctate dots in Bowman's zone in a case two weeks following the accident. There was little conjunctival reaction at the time.

sequelae may follow. A particle of lime, if retained, may cause continued destruction with formation of a localized dense scar. Lime burns tend to produce deformities (pseudopterygium) of the eyelids and conjunctiva, which in themselves may cause corneal complications. A typical picture of recurrent epithelial erosion may follow prolonged exposure to chemical vapors or dust.

War gases, such as mustard gas or phosgene, produce considerable

corneal damage, depending on the concentration and time of action. The damage may vary from a slight vesiculatory epithelial necrosis to parenchymal destruction with subsequent scar formation. Indolent or recurrent corneal ulceration may persist for many years following mustard gas burns.

Rare instances of corneal ulceration have occurred, following the use of hair dyes and cosmetics about the eyes.

Superficial burns of the cornea may be caused by agents such as ether, tincture of iodine, depilatories containing barium sulfate, and many other toilet preparations. Some of these cause marked chemosis of the conjunctiva and superficial corneal edema. Permanent damage seldom results from accidental burns by these substances, although tiny subepithelial (Bowman's zone) dots may be visible for some time.

Insect stings, if not promptly removed, may cause a severe corneal inflammation owing to their formic acid content.

The cornea may be damaged by the action of short wave (ultra-violet, roentgen ray and radium) or long wave (infra-red) radiation. An abiotic reaction follows exposure to short wave radiation, whether solar or from industrial or therapeutic apparatus. Varying with the intensity and frequency of exposure, this reaction appears after a latent period of from six to eight hours. It is characterized by edema of the epithelium which, if severe, may result in vesicle formation and desquamation. In extreme lesions, the parenchyma may be involved, with the formation of a deep opacity, caused by lamellar coagulation.

Long exposure to ultraviolet radiation (as in the case of welders, persons with snow blindness, and the like) may produce band-shaped opacity in the anterior corneal layers corresponding to the interpalpebral zone. The cornea may or may not become vascularized. A hazy, semitransparent scar may result.

Infra-red radiation (heat rays) causes a thermal lesion in the cornea. In this case the parenchyma first becomes uniformly opaque, with destruction of the endothelium; the epithelium, however, may remain normal. In extreme cases there is permanent scarring.

Chapter Sixteen

THE ANTERIOR CHAMBER

BIOMICROSCOPY has made possible satisfactory examination of the anterior chamber. Not only can its boundaries be studied minutely but its contents as well.

Anatomically, the normal anterior chamber is bounded in front by the endothelial lining of the posterior corneal surface. Its deep boundary consists of the anterior surface of the iris, and, depending on the size of the pupillary area, by the anterior lens capsule. An acute angle is formed by the meeting of the inner surface of the cornea and the base of the iris, ciliary body, and trabecular tissue at the corneoscleral junction. The endothelial lining of the anterior chamber is continuous over the posterior surface of the cornea, incorporated in the angle trabecula and eventually passes over the iris, being interrupted at the crypts and at the pupillary margin.

According to Salzmann,²⁶² the vertical and horizontal diameters of the normal anterior chamber are about equal and vary, with the individual, from 11.3 to 12.4 mm. The depth of the normal anterior chamber varies according to the age of the individual and the refractive condition present. The depth of the anterior chamber is greatest in the pupillary zone and diminishes gradually toward the periphery. Because of the abrupt thinning of iris stroma at the beginning of the iridocorneal angle, the depth at this point may be less than at the more peripheral portions in the angle itself. Raeder,²⁴⁶ employing a special technique, substantiated the conclusions of other workers that the depth of the anterior chamber in emmetropic individuals decreases with age. Between the ages of 15 and 65 the average axial depth of the anterior chamber decreased from 3.69

to 3.04 mm. Therefore, it is only possible to give an average chamber depth for a given age. This decrease in depth, associated with aging, is attributed to an increase in the thickness of the lens.

The chamber is shallower in hyperopic than in emmetropic individuals of the same age. In myopia, the anterior chamber is deeper than it is in emmetropia. Practically, the approximate depth of the anterior chamber can be judged by the width of the dark intervening space between the posterior surface of the corneal parallelepiped or section and the anterior surface of the iris or lens. The Ulbrich drum may be employed to obtain a more accurate relative measurement of this distance by means of microscope displacement (page 119).

In measuring the depth of the chamber, the axis of the microscope should be perpendicular to the cornea. In this position the apparent difference in depth between two fixed points may be measured. To obtain an absolute value, a correcting factor must be employed for corneal refraction. As the angle between the axis of observation and the axis of the beam is increased, the dark intervening space (anterior chamber) becomes geometrically greater. This fact is helpful in studying the contents of a shallow anterior chamber since the intervening space traversed by the oblique beam between the iris and the cornea is enlarged because of the longer path of the beam through the anterior chamber.

TECHNIQUE OF BIOMICROSCOPIC EXAMINATION OF THE ANTERIOR CHAMBER

For practical purposes, the anterior chamber of the normal eye is optically empty and dark. As a result, when the critically focused beam traverses the aqueous, there is a dark space between the illuminated cornea and iris or lens (Fig. 41). However, in special cases, when the eyes of the observer are dark-adapted and high intensity illumination (arc lamp) and a special technique are used, it is just possible to perceive a faint Tyndall flare in the normal aqueous. This is due to the fact that the albuminoid content of the aqueous is 20 mg. per cent. For example, when a cylindric beam

passes through the normal aqueous, it may be just possible to see (with the dark pupil as a background) that the unilluminated portions surrounding the beam are somewhat darker than the aqueous in the direct path of the beam. The appreciation of a Tyndall or aqueous flare depends on the luminescence of the beam in contrast to its dark surroundings. The amount of luminescence is a function of the colloidal content of the aqueous and the intrinsic brightness of the slit beam. Therefore, if the beam is constant in brightness the variations in luminosity of the aqueous section are indicative of a varying protein content of the aqueous. Furthermore, the degree of darkness of surrounding aqueous, which provides a contrasting background (on which the visibility of the beam also depends) may be modified by light scattered or reflected from the iris or lens. Absence of ideal conditions (clear cornea or black pupil) adds to the difficulty in securing the necessary dark contrasting background; such interference may be caused by a hazy cornea, cataract, small secluded pupil, or by a very shallow anterior chamber. These or similar complications make the detection of a Tyndall flare quite arduous, unless it is marked.

Kronfeld¹⁸⁴ endeavored to determine the minimal protein concentration that could be recognized as a definite flare in eyes in which the conditions present were not ideal for the recognition of a faint beam. His method consisted of first determining the visibility of an aqueous flare by comparison with its surroundings, and then determining the protein content by aspirating the aqueous. He found that the average for the visibility threshold appeared to be about 230 mg. per cent (ten times as high as the normal aqueous protein content). This would indicate that increases of protein content up to ten times the normal aqueous protein may cause no appreciable difference in luminescence of the light path in the anterior chamber.

Roenne²⁵¹ in devising his colloidometer (as an attachment to the Zeiss-Gullstrand biomicroscope) ingeniously utilized the corneal section as the standard for comparison with the luminosity of the aqueous flare, thus obviating the necessity of depending on the

variable contrasting background in the older method over which the observer has no control. This was the first instrument devised for quantitative tyndallimetry of the anterior chamber.

The colloidometer consists of a set of twelve neutral gray (Tscherning) filters in a Rekoss disk placed in relation to the slit opening, so that each filter can be rotated to cover the lower four fifths of the slit, leaving the upper one fifth uncovered. In this way the intensity of illumination of the lower four fifths of the corneal section may be varied by rotating the suitable filter into the slit until the luminosity of the corneal section (lower four fifths) is equal to the upper aqueous section (upper one fifth) which receives the full intensity of the beam. The filters vary in transmission from $1/320$ to $1/3200$ (Figs. 254, 255). The filter $1/320$ has been found to represent the upper limit of normal.

When making a measurement, the gray filter reduces the luminosity of the lower four fifths of the corneal section until it matches that of the unfiltered upper one fifth of the aqueous section. The transmission factor of the gray filter measures the protein content of the aqueous.

The ability of the instrument to provide a series of gray filters, covering the entire range of luminosity of the aqueous sections, permits selection of an appropriate corneal section for comparison with any degree of aqueous luminescence. This tends to facilitate recognition of very low degrees of aqueous flare which, using the older methods, are recognizable only with difficulty.

Kronfeld and his associates correlated the protein content of the aspirated aqueous as determined by chemical analysis with the colloidometric readings in six cases and demonstrated a fair parallelism. Furthermore, by using a contact glass and filling the intervening space with a serum dilution of suitable strength they were able to perform tyndallimetry of the cornea itself. They also showed that the corneal section may vary in luminescence, a factor which may have to be considered in accurate work.

In order to perceive the flare or relucency in the normal aqueous, it is advisable to have a sufficient field of observation so that a com-

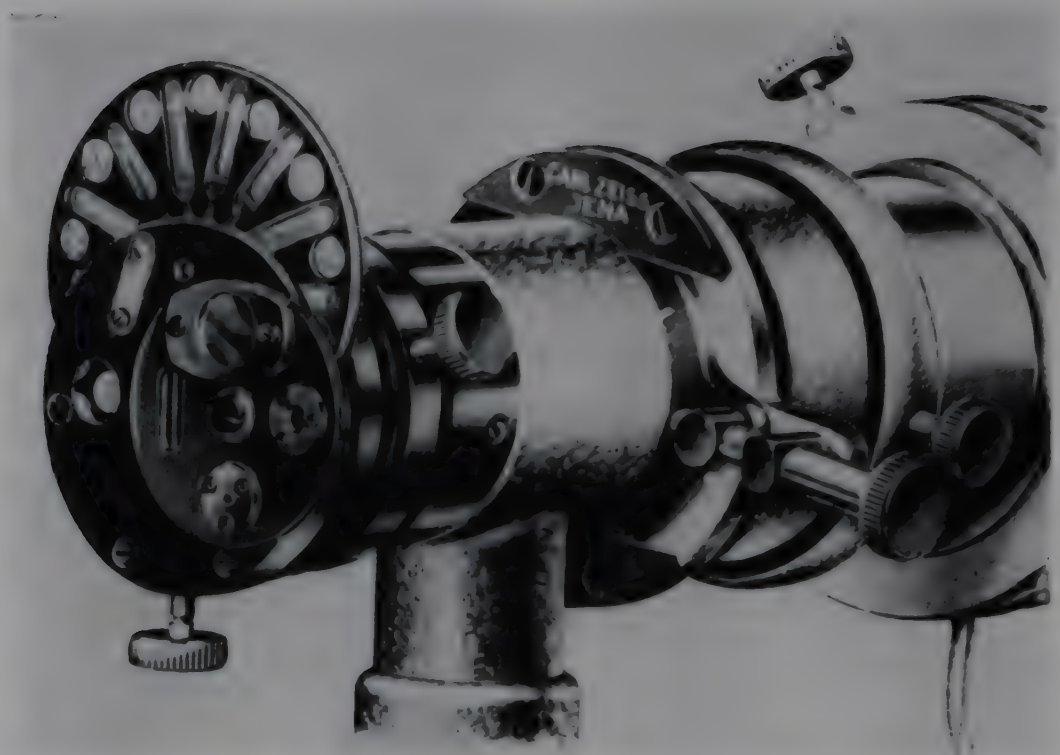


FIG. 254. Roenne's colloidometer. (Courtesy of Carl Zeiss, Inc.)

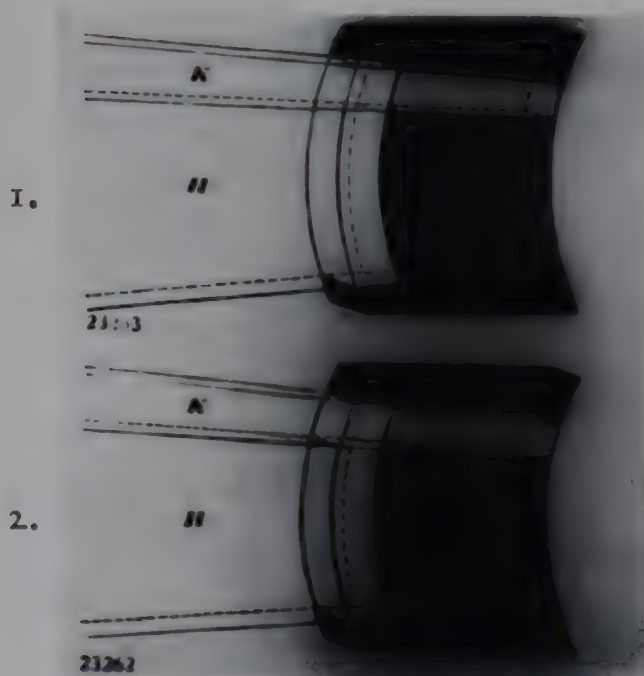


FIG. 255. Diagram illustrating the use of the Roenne colloidometer. 1. The passage of the light beam in the cornea and anterior chamber. The lower four-fifths (H) has passed through a gray filter, the upper one-fifth (K) is unfiltered. In upper figure, the unfiltered light (K) in the cloudy anterior chamber is darker than the portion (H) in the cornea. In other words the gray filter is not dark enough.

2. In the lower figure the correct filter is used because the paths of light in the anterior chamber (K) and in the cornea (H) are equal in intensity.

parison can be made of the differences in luminosity between the portions of the aqueous actually traversed by the luminous beam and the adjoining portions. This requires low magnification. How-



FIG. 256. Method of obtaining the normal flare of the anterior chamber, demonstrating the lowered position of the beam to allow comparison between the dark pupil above (at point indicated with arrow) and the flare in the anterior chamber. The retro-illuminated light from the iris also helps to accentuate the flare.

ever, with higher powers the small cylindric beam can be used to obtain the same effect.

When the optic section is utilized, a contrasting effect can be obtained by directing the luminous beam in such a manner that the upper portion of the beam partly enters the lower part of the pupil. This permits the dark areas immediately above to serve as a contrasting background (Fig. 256).

In view of the difficulty involved in demonstrating a flare in the normal aqueous, for practical purposes the presence of an appreciable flare is indicative of a pathologic state.

A definite flare always indicates a pathologic increase in albuminoid content. It should be pointed out that a flare may be obtained without the presence of formed elements (e.g., cells, fibrin, coagulates), but in most cases these elements are soon observed or are present from the beginning. Demonstration of a Tyndall phenomenon in the aqueous demands concentrated focal illumination,

such as that afforded by direct focal illumination. However, for large structures within the aqueous (e.g., cysts, foreign bodies, membranes) or for changes on the bounding surfaces (especially on the posterior corneal surface) diffuse or retro-illumination and specular reflection are of definite value. Using the endothelial specular reflex as a point of reference, it may be possible to judge whether or not structures in the anterior chamber are in actual contact with the cornea.

A strongly concentrated intense focal beam, such as is obtained with an overloaded lamp or carbon-arc source, is of advantage in studying the contents of the chamber. Naturally, the examining-room should be dark and the eyes of the observer dark-adapted. Specular reflection must be avoided (except when the corneal endothelium is under observation) because of the interference that is produced. This can be accomplished by having the patient move his eye or by altering the angle between observation and illumination. Narrowing of the beam decreases interference from specular reflection but has the disadvantage of diminishing the intensity of illumination. The cylindrical beam, when used with a high intensity light source, gives the best contrasting effects but because of its small size, requires more manipulation in order to cover the entire anterior chamber.

Adequate examination of the anterior chamber demands that the observer vary the incidence, intensity, and size of the luminous beam to suit the local conditions. As the beam traverses the anterior chamber from the temporal side, it either enters the pupil and passes into the lens or it falls on the iris. To obtain a large area of dark pupillary background, the beam is allowed to strike the nasal side of the iris. The microscope should be focused deeply in the anterior chamber to the nasal side of the corneal parallelepiped, using the dark pupil as a background. When feasible, partial mydriasis is advantageous to increase the area of the dark background. Directing the beam from the nasal side, that is, across the nose, enhances observation of the temporal portions of the chamber. Vogt recommends that in searching for aqueous floaters, the area adjacent to

the illuminated iris be used because of the slight increase of illumination obtained by the reflections from this area. He terms this zone the "optic border zone."

In certain instances, for purposes of orientation and to ensure accurate focusing of the beam, it is well first to focus the microscope on the posterior face of the parallelepiped and then to rack it forward and somewhat laterally, so as to focus deeper into the aqueous to one side of the parallelepiped. In this way, the aqueous, which lies along the pathway of the luminous beam between the parallelepiped and the illuminated iris or lens, is examined.

In addition, fine vertical or horizontal oscillatory movements may be made with the beam in order to enhance the effect of contrast. Also, movement of the eye by the patient may cause motion of suspended particles or strands.

THERMAL CONVECTION CURRENTS IN THE AQUEOUS

Owing to the difference in temperature which exists between the deeper aqueous (warmed by the vascular circulation of the iris) and the anterior aqueous, lying in proximity to the avascular cornea (cooled by the external atmosphere), there is a temperature gradient, especially in the area corresponding to the palpebral opening. The difference in temperature between the cornea and the iris has been estimated as ranging from 4 to 7° C. and in consequence convection currents in the aqueous are formed. These arise because of the difference in density which occurs between the warmer and cooler aqueous.

With the biomicroscope, convection currents are not visible in the aqueous unless floating cells or particles are present. Even when there is a marked Tyndall effect (resulting from an increase of albuminoid content), no current is discernible because of the sub-microscopic size of the protein molecule.

Only the movements of particles carried by the thermal currents indicate the course of these currents. Normally, in children, cells (leukocytes) may be present physiologically in the aqueous, and in the aged occasional pigment granules are discernible. As in patho-

logic states, these may reveal thermal convection currents in the normal eye.

Because the anterior chamber is deeper in the pupillary area, a greater concentration of floating cells is visible in this zone. For this reason, convection currents are studied best in this region. It should be remembered that when physiologic cells are present, little, if any, increase in aqueous flare is visible.

In view of the temperature gradient (which may be influenced also by the heating effect of the focal beam), the aqueous currents pursue vertical directions in which they rise in front of the iris and descend behind the cornea. This fact is proved by the visible movements of suspended cells, which rise in the deeper aqueous and fall in the anterior portions behind the cornea. The downward movement in the central pupillary area is in a straight line. This may be contributory in the formation of the Ehrlich-Turk line because the inward curving of the cornea intersects the vertical downward course of the current (page 429).

The deeper the particle lies in proximity to the plane of the iris, the faster it rises. This is best seen in the pupillary area where the ascending particle attains its maximum speed (2 mm. in from two to four seconds). The more laterally situated particles rise in a curvilinear fashion to the sides and then proceed downward following the curve of the cornea (Fig. 257). According to Erggelet, the stream becomes slower inferiorly in the area where it turns toward the iris to rise. It may swirl about at this point or stagnation may occur, depending on the position of the eyes, direction of the focal beam, and associated factors. Apparently the heat of the focal beam can induce the formation of swirling eddies.

Plocher observed that in the lower areas of the anterior chamber, depending on the number of the particles and the position of the eyes, that the particles remained at a distance from the cornea and had a tendency to move in an elliptical stream backward or to curve to the sides and then proceed directly upward in front of the iris. When the focal beam is permitted to remain fixed on one portion of the cornea, a heating effect ensues, which causes the descend-

ing currents to slow. Berg established the thermal basis of the existence of the convection currents. He noted that a change in position of the patient's head did not alter the vertical direction of the



FIG. 257. Directions of convection currents in the anterior chamber. Heavy lines indicate the rising movement in deeper part of anterior chamber; finer lines indicate direction of currents in anterior part of chamber. (After Meesmann.)

current and that bathing the cornea with warm saline solution (thus equalizing the temperatures in the anterior chamber) caused slowing of the current and eventual cessation or even reversal. Plocher noted that when a patient had reclined with closed eyelids for a period of time, about ten minutes was required for the current to become re-established after the eyes were opened and a vertical position reassumed. The cessation of the convection current which occurs under these circumstances is probably due to the warming effect of the eyelids closed over the cornea and to altered gravitational direction in the supine position.

Viscosity is another physical factor which influences the characteristics of the current. As the albuminoid content of the aqueous rises pathologically, its viscosity increases and a corresponding slowing of the convection currents is noted, in conjunction with a marked increase in aqueous flare. At the height of an inflammatory process the cellular elements will be seen as if tremulously suspended in a gel. According to Meesmann an increase of albuminoid content to 2 per cent arrests the convection currents. The practical importance of this is demonstrated by the fact that during the course of progressive iridocyclitis, the velocity of the convection current (as seen by cell movements) decreases as the aqueous flare becomes more marked. As regression occurs and the albuminoid content of the aqueous diminishes, the flare becomes less marked and the velocity of the convection current increases. Observation of these phenomena assists in determining the progress of such inflammations.

Large fibrinous coagulum (strands, gelatinous masses), because of their weight and propensity to early attachment to the endothelial-lined boundaries of the anterior chamber, exhibit movement in the convection current only to a limited degree, if at all. This likewise holds true for pupillary rests and glass membranes.

CONGENITAL ANOMALIES OF THE ANTERIOR CHAMBER

ABNORMALITIES IN DEPTH OF THE ANTERIOR CHAMBER

As previously mentioned (page 558), variations in depth of the anterior chamber usually occur in association with increased age and with changes in the refractive state. Congenital variations of depth are generally associated with other ocular abnormalities.

In *megalocornea* and *buphthalmos* there is considerable deepening of the anterior chamber. In an analysis of 20 buphthalmic eyes, Gross¹³⁸ found the average depth to be 6.3 mm., the normal average being taken as 2.6 mm.

Megalocornea (or anterior megalophthalmos³¹³) is due to a primary overgrowth of the cornea; however, in buphthalmos, secondary pressure — stretching of the cornea, owing to the changes in the angle — leads to secondary enlargement of the anterior chamber.

Analogously, microcornea represents an undergrowth of the cornea unassociated with other changes, while microphthalmos (in which a shallow anterior chamber also occurs) is generally accompanied by other deformities (e.g., colobomas, cysts). In some eyes, the iris may be completely adherent to the cornea or it may be absent altogether. In the former case there may be no anterior chamber.

In a case already described (page 299), there was a microphthalmic eye with broad anterior iridic synechiae traversing the shallow anterior chamber and attached to a hyaline membrane on the posterior corneal face.

Various types of membranes and pigmented bodies can be found in the anterior chamber. These are: persistent pupillary membranes, membranes following fetal iritis, congenital anterior synechiae, and hyaline membranes. A partially detached ring of pupillary flocculi has also been described by Dymschitz.⁷⁵ Completely detached cystic flocculi may become free in the anterior chamber, appearing as small brownish spheroids. Occasionally a strand of attachment to the pupillary margin is observed. The cystic varieties probably originate as detached flocculi. Increase in the size of these cysts has been noted in some instances, and collapse with subsequent reformation in others. Clapp⁴⁶ reported a case of a free cyst in the right eye, which was seen as a dark oval body (0.75 by 1 mm.). It was freely movable. Ordinarily situated below, the cyst gravitated to the dependent center of the cornea when the patient was placed in a prone position with his face down. With the biomicroscope the surface of the cyst was seen to be unevenly covered by pigment granules on a delicate basement membrane, the interior being filled with a clear substance.

PUPILLARY MEMBRANE

Pupillary membranes may also be considered as anomalies of the anterior chamber. They may appear in a variety of forms. Complete persistence of the fetal pupillary membrane is sometimes seen, especially in microphthalmos. The pupil is entirely covered by a dense matlike membrane. In the newborn the circulation can be

traced but later the vessels turn into white strands or lines, frequently pigmented. The usual form of pupillary membrane is that of an incomplete persistence. This may occur as single or multiple strands with or without adherence to the lens capsule. These remnants of the pupillary membrane usually cross the pupil and are attached at both ends to the lesser vascular circle by weblike expansions. In some instances two or more fibers may leave the lesser circle and then unite in the pupillary zone or at their extremities in a festoon-like fashion. The fact that most of the remnants of the pupillary membrane terminate in the lesser vascular circle serves to differentiate them from membranes of purely inflammatory origin, which in most cases are connected to the pupillary border.

Occasionally, in iris coloboma, persistent capsulo-pupillary vessels from the lesser vascular circle of the iris may be seen extending behind the lens. Frequently, these pupillary remains may be attached to the lens capsule; such lenticular attachments may be marked by a capsular opacity or a stellate pigment deposit or, sometimes, by an anterior pyramidal cataract (Plate XXXVIII, figs. 1, 2). At times loose, unattached ends float freely in the aqueous. Other forms of pupillary membrane may result from the projection of iris stroma or vascular loops beyond the pupillary margin into the pupillary area (Fig. 258).

Fetal iritis is responsible for several types of pupillary membranes. The appearance depends on the stage of development of the fetal pupillary membrane at the time of the intra-uterine inflammation. When the inflammation occurs while the pupillary membrane is in existence (i.e., before its resolution), posterior synechiae develop between the engorged vessels and the lens capsule and between the pupillary margin and the capsule. The final appearance comprises a more or less irregular pupillary membrane usually with lens adhesions either with or without associated posterior synechiae. Later (i.e., after the formation of the lesser circle) the remaining inflammatory vessels may assume a looped pattern not unlike that seen in persistent noninflammatory vessels.

It must be remembered that in fetal iritis, new vessels may form

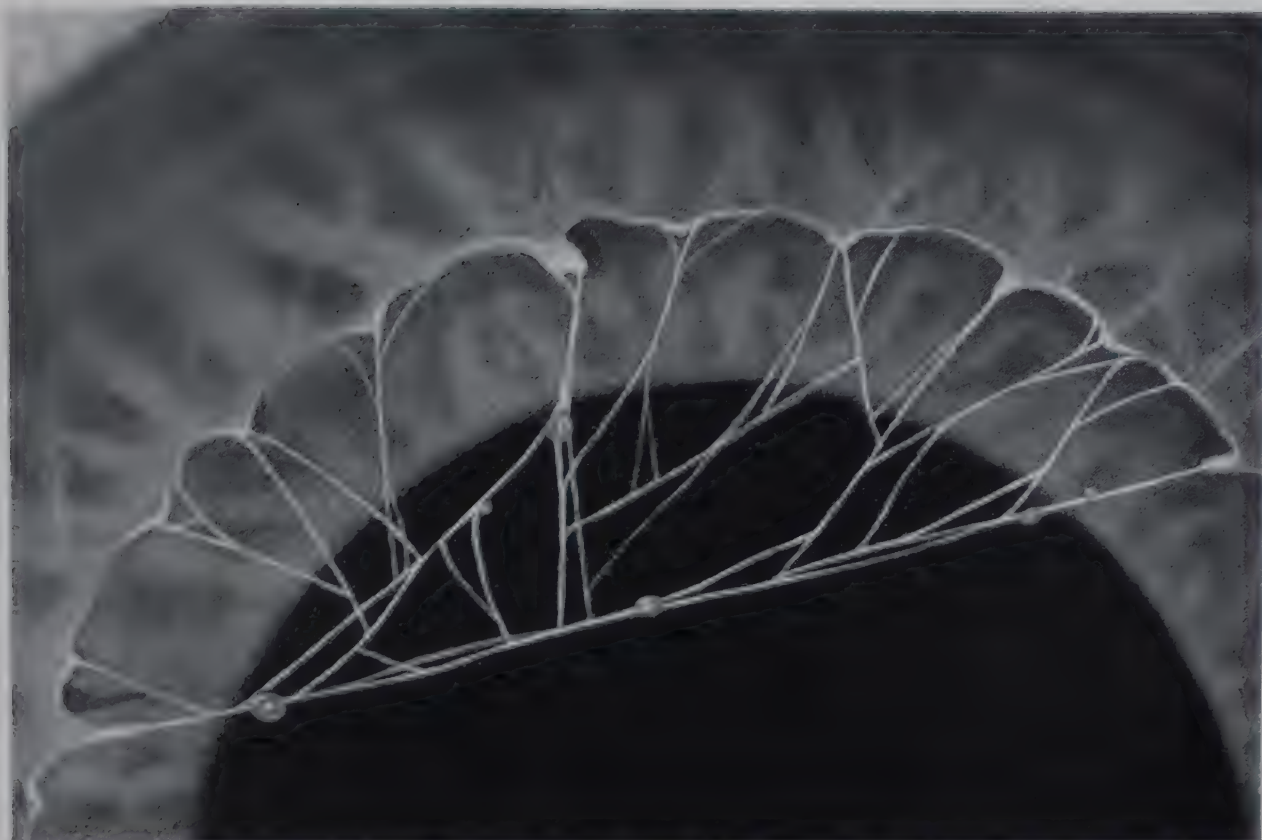


FIG. 258. Persistent pupillary membranes extending over pupillary area.

either the lesser vascular circle or the pupillary margins. Pigmented deposits on the strands of all types of persistent membrane are frequently seen.

As a result of disturbances of mesodermal development various types of *congenital anterior synechiae* and *hyaline membranes* may form (Plate XXXVIII, figs. 3, 4). The synechiae may extend from the lesser vascular circle to the cornea (page 299) or they may be peripherally situated. Occasionally, they are associated with hyaline membranes which usually are attached to the posterior corneal surface and may appear as a peripheral retrocorneal ring. It is still unknown whether these ring-type peripheral or central plaque hyaline membranes are due to maldevelopment of the cornea or of the iris. Hyaline membranes, some of which traverse the anterior chamber and are attached to the posterior corneal surface, have been reported in association with other congenital anomalies, such as eyelid colobomas, epibulbar dermoid and dermolipoma, iris coloboma and corectopia.

ALTERATIONS IN THE ANTERIOR CHAMBER FOLLOWING TRAUMA *

Trauma to the eyeball, even though slight, may cause immediate alterations in the anterior chamber, visible with the biomicroscope. It is, therefore, important to examine all injured eyes as soon as possible in order to be able to evaluate future changes, which may be significant from a medicolegal standpoint.

Even mild contusions or the presence of a foreign body on the cornea may evoke reactions in the anterior chamber. More severe contusions or perforating injuries are always accompanied by an aqueous flare due to an outpouring of cellular elements and albumin (fibrin). This exudation has also been compared with a fine dust and is composed of the corpuscular elements of the blood, and their derivatives (liberated pigmented granules). Such debris may be completely absorbed within a few hours or days. This point is of value in differentiating traumatic from inflammatory exudates which persist for a longer time.

* The changes occurring in the so-called "traumatic anterior segment syndrome" and in sympathetic ophthalmia are considered in Volume II.

Cases have been seen (Vogt) in which opaque clouds with admixed cellular elements in varying degrees appeared in the aqueous within an hour following injury. Within from sixteen to eighteen

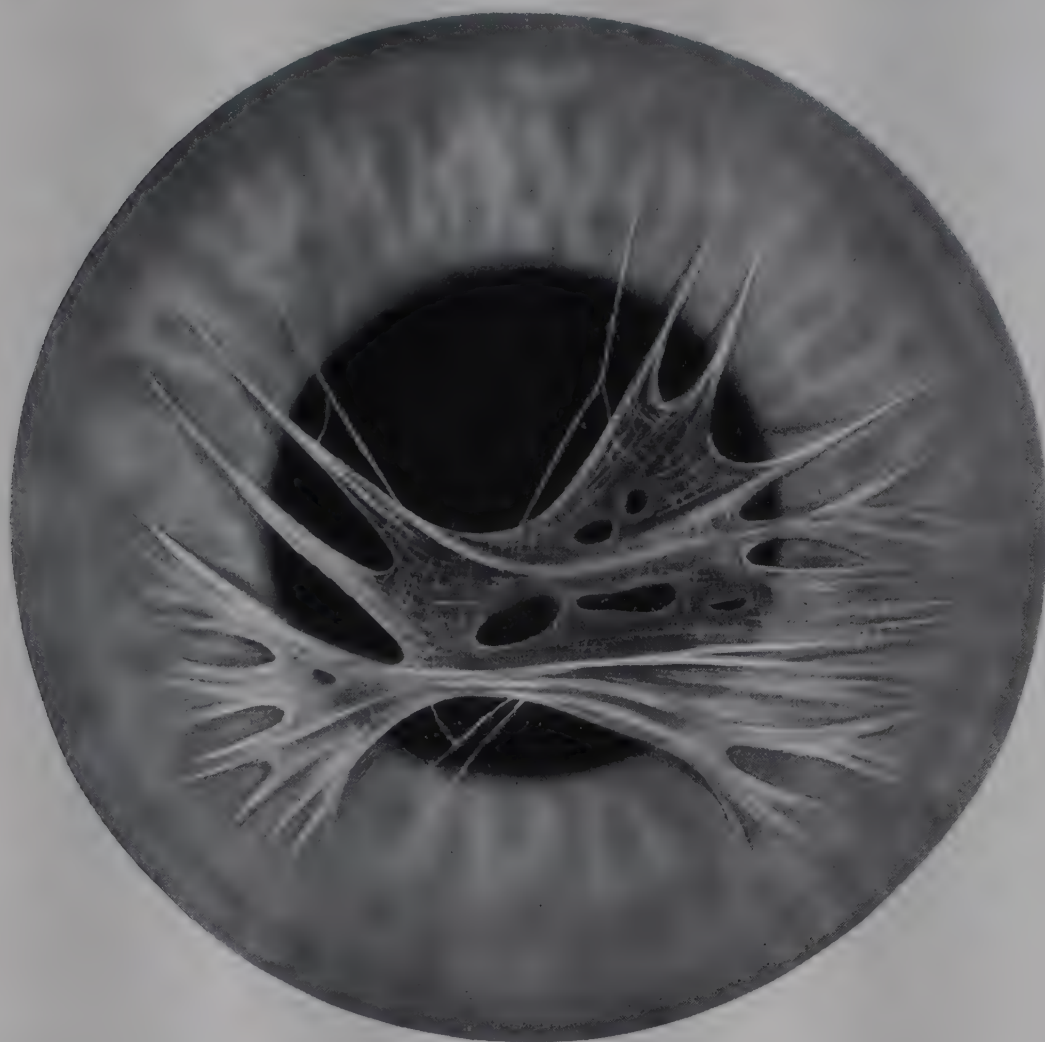


FIG. 259. Fibrinous coagula in the form of pupillary membranes seen one hour after contusion to eye.

hours almost complete absorption occurred except for one or two white dots in the anterior chamber. The presence of cells in the anterior chamber leads to the demonstration of convection current and the formation of an Ehrlich-Turk line. Red blood corpuscles may be recognized by their glistening and pale yellow color. Fibrinous coagulum may likewise appear during the first hours after a contusion or perforation; it may form solid masses or delicate threadlike designs with or without pigments. When attached to the lesser arterial circle, this coagulum may resemble a pupillary membrane (Fig. 259). Within a few days such structures may be entirely absorbed. Fibrin-like masses are more likely to occur following perforating injuries than after slight contusions. In the latter case one is more likely to see small coagulated fluffs surrounding

PLATE XXXVIII

FIG. 1. Persistent pupillary membranes. Diffuse illumination. The membranes are attached to the anterior lens capsule, the surface of which is marked by the presence of stellate pigment remains.

FIG. 2. Details of the attachment of these membranes (Fig. 1) to the lens. Direct focal illumination (high power).

FIG. 3. Hyaline membrane of posterior corneal surface with attached anterior iritis membranes in a case associated with syndactylia.

FIG. 4. Same case as in Figure 3. Details seen with optic section. One of the membranes is seen attached to the posterior surface of the corneal section.

FIG. 5. Glass membranes in the anterior chamber following birth trauma in direct focal illumination; attachment of the extremities of the membranes to the posterior corneal surface is seen below. These membranes (cordlike) bend backward in the anterior chamber.

FIG. 6. A case of extensive persistent pupillary membranes. The membranes extend from the iris frill to the anterior lens capsule where their expansions together with pigment remains form a denser opacity.



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suspended cells in the pathway of the gray-appearing relucant beam. The interesting feature of these changes is the rapidity with which they appear and resolve. Extensive ruptures of the iris may be attended by considerable hemorrhage leading to a gross hyphema, which may or may not absorb quickly.

Shrinkage and absorption of a hyphema usually begins in the portions contiguous to the posterior corneal surface so that an empty space may first appear between the cornea and the coagulated blood. When the beam is directed above the hyphema a positive Tyndall phenomenon is usually revealed owing to the presence of yellowish brown blood elements. Deposits of these elements may be seen on the posterior corneal surface. A curious arrangement of red blood corpuscles on the posterior corneal surface in the form of parallel vertical columns extending from the hyphema toward the pupillary area has been observed (Plate XXXI, figs. 1, 2). Such columns, which may number from two to ten, may be transient and change or even disappear rapidly. These formations are readily seen by retro-illumination; they are composed of fine droplike cells of reddish hue. The vertical appearance of the linear deposits suggests that seen in an Ehrlich-Turk line.

One consequence of a contusion (even though uncomplicated by gross damage or perforation) is the development of ocular hypotony. Although usually preceded by post-traumatic hypertension, marked reduction of tension soon follows in most cases of concussion or contusion of the globe. Ocular hypotony may also follow prolonged bandaging or massaging of the globe. Duke-Elder⁷³ believes that a local nervous axon-reflex mechanism causes sufficient disturbance of the ocular circulation as to lead to hypotony. This is manifested biomicroscopically by a demonstrable increase in depth of the anterior chamber.

Following perforation of the cornea (either surgical or traumatic) a great variety of alterations of the anterior chamber may occur, depending on the nature and extent of the insult.

In addition to the aforementioned exudation into the aqueous, lacerated tissue may be separated from the cornea, iris or lens and

be deposited in the anterior chamber (Plate XL, fig. 1). Such tissue may be either free or attached more or less completely to the damaged surface or it may be adherent to other structures (e.g., a detached



FIG. 260. Spontaneous rupture of Descemet's membrane. [After Lloyd.]

shred or plaque of Descemet's membrane may be seen adherent to the iris or lens or may be found free in the lower angle of the anterior chamber). Vogt described a spear-shaped hyaline fragment of Descemet's membrane, extending from the posterior corneal surface into the anterior chamber. This occurred following a discission.

GLASS MEMBRANES

Many authors^{85, 126, 135, 211} have reported cases in which the formation of glasslike hyaline membranes in the anterior chamber were associated with ruptures of Descemet's membrane spontaneously (Fig. 260) or following birth injuries (Plate XXXVIII, fig. 5). Such membranes may be single or multiple, running parallel to

each other or radiating in fanlike bands, and at times actually partitioning the anterior chamber. These membranes may be attached to the posterior surface of the cornea at one end only. In

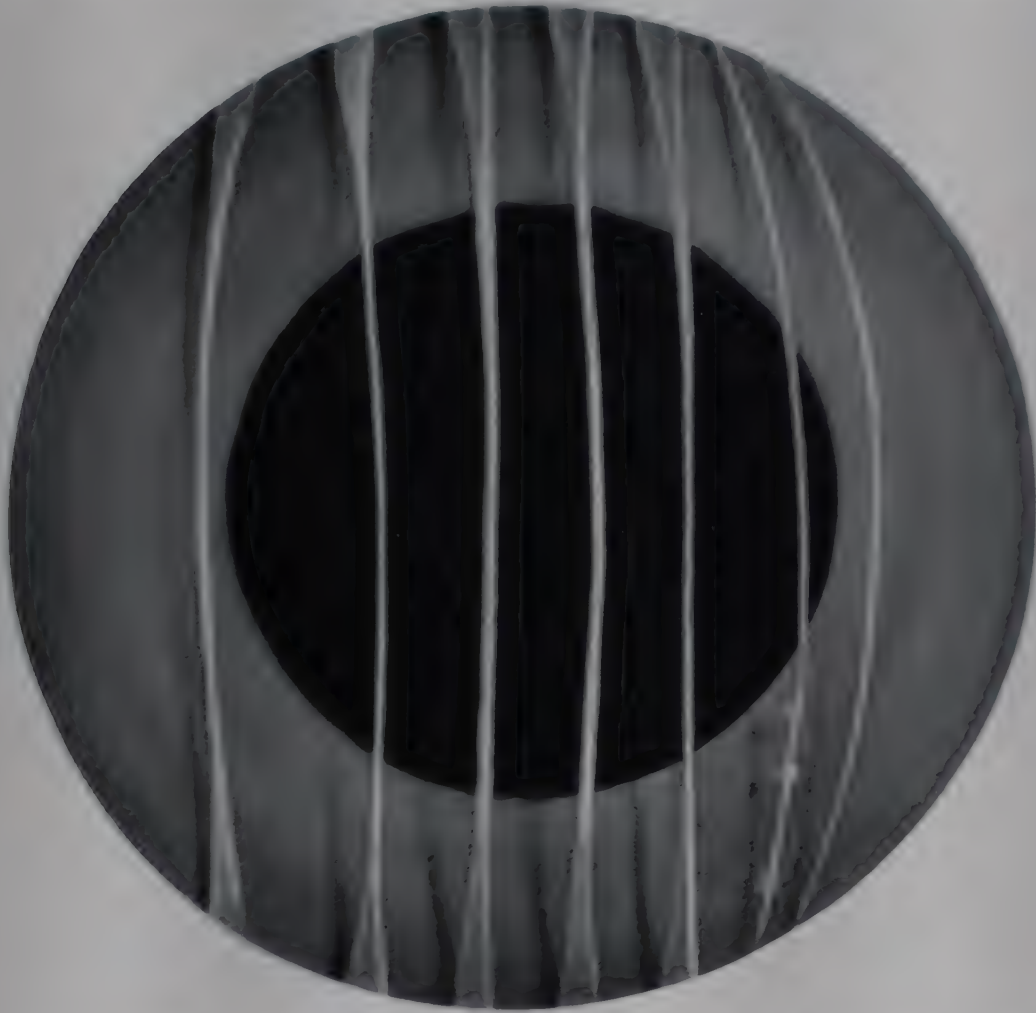


FIG. 261. Glass membranes in the anterior chamber following birth injury. (After Lloyd.)

this case, the free end in the anterior chamber usually has a curled edge. In other instances parallel stripes or bands extend across the anterior chamber attached at each extremity to the posterior surface of the cornea (Fewell's case). Lloyd¹³⁶ described a case illustrating such a formation (Fig. 261). The patient was a male, aged 18, with a history of forceps delivery. The right eye was normal; vision in the left eye was 2/200 unimproved by means of glasses. The deeper layers of the cornea were cloudy. All the strands except the first were free in the anterior chamber and bore a strong resemblance to a bow string. In another case of Lloyd's, there were numerous tears in Descemet's membrane, occurring in the left eye with reduction of vision to 10/200. A history of birth injury by forceps was elicited. There was keratoglobus and a deep anterior

chamber. Peters²³⁶ made a histologic examination of the eye of a child who died twenty-three days after delivery by means of instruments. This revealed that the detached strips of Descemet's membrane curled up laterally and that the endothelium covered both sides of the strip. During life the deeper layers of the cornea were cloudy, owing to aqueous seepage into the parenchyma through the rents in Descemet's membrane.

The changes produced by traumatic lacerations of Descemet's membrane are usually permanent. As part of the repair process the endothelium may secrete a new glass membrane. I reported²³ a case of elaboration of such membranes which led to the formation of a cyst on the posterior corneal surface. The gap became filled with a new hyaline membrane secreted by the adjacent endothelial cells.

CYSTS OF THE ANTERIOR CHAMBER

Cysts of the anterior chamber may result from implantation of the surface epithelium traumatically or postoperatively, from reduplication of Descemet's membrane after tears (page 422), from iris or ciliary body sources, and finally from parasitic invasion of the anterior segment. Rarely, cyst formation on a neoplastic basis has been observed. Pearl cysts arising from implantation of cilia or integumentary epithelium appear as firm whitish tumors on the iris.

Implantation cysts, probably the most common type of cyst, usually occur postoperatively. The ingrowing or implanted epithelium tends to grow along the walls of the anterior chamber and, when blocking the filtration angle, to cause ocular hypertension. With the biomicroscope the epithelium appears as a fine filmy veil on the posterior surface of the cornea. In optic section, the existence of a double line may sometimes be demonstrated. In the retrocorneal cysts, which follow tears in Descemet's membrane, the appearance of a double layer caused by the reduplication of the glass membrane is seen.

Slow reformation of the anterior chamber following operation associated with slow healing of the wound and the development of anterior chamber fistulae should lead one to suspect the presence

of epithelial implantation. In Vail's monograph³¹⁴ on epithelial downgrowth in the anterior chamber following cataract extraction, there are several biomicroscopic descriptions. The main points brought out are that a connection of the epithelial downgrowth with the corneal wound could be established and that, owing to improved nourishment, a more rapid growth of the epithelium occurs on the iris than on the posterior corneal surface. In addition Vail points out that the filmy membrane is more easily discernible over a brown iris than over a blue or gray one. In a case reported by Custodis there were little pearls which resembled air bubbles; these were probably due to degenerative cell changes. Cyst formations must be differentiated from reticular membranes, inflammatory membranes, and proliferated capsular elements. It should be emphasized that in cases of glaucoma following cataract operations biomicroscopic examination should always be made to exclude the possibility of epithelial ingrowth into the anterior chamber.*

PARASITES

Cysticercus cellulosae has been observed to invade the anterior chamber. It appears as a globular translucent cyst in the anterior chamber, usually attached to the iris and more rarely to the anterior lens capsule. Movements of the parasite can be observed. Unless it is removed, severe plastic iritis may result.

VITREOUS PROLAPSE INTO THE ANTERIOR CHAMBER

Prolapse of the vitreous into the anterior chamber generally follows intra-ocular surgery or severe contusions or perforating injuries in which tearing of the zonular ligaments is a prominent feature (Plate XXXIX, fig. 4). In all cases of contusion followed by persistent increased ocular tension, this complication should be suspected.

The only objective finding in such injuries of the zonule may be a bead of vitreous prolapsed into the anterior chamber, even though frank luxation of the lens cannot be proved. However, large

* The various forms of cysts of the iris are discussed in Volume II.

PLATE XXXIX

FIG. 1. Mooren's ulcer of cornea. Diffuse illumination.

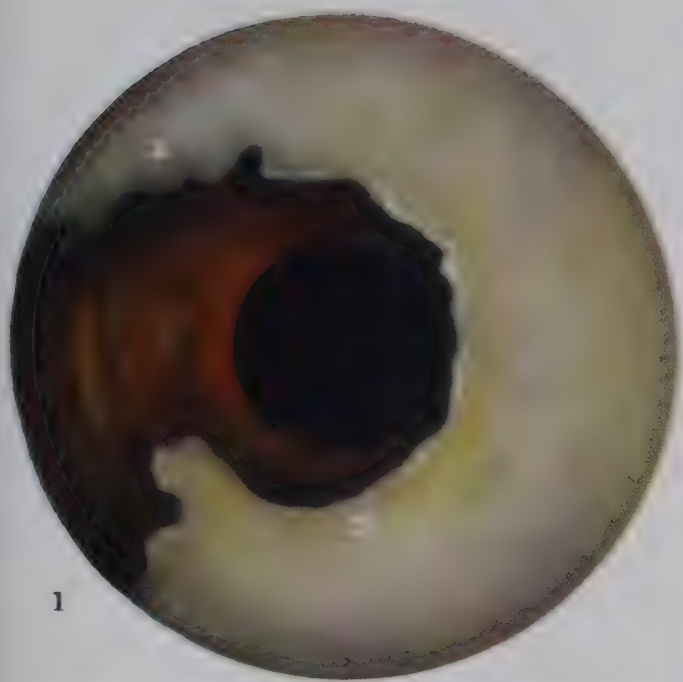
FIG. 2. Mooren's ulcer. Details at upper edge of ulcer. Shown in Figure 1. Folds in Bowman's membrane.

FIG. 3. Mooren's ulcer. Details at lower edge of ulcer.

FIG. 4. Aphakia. Vitreous and some lens material adherent to perforating corneal scar (traumatic).

FIG. 5. Implantation of cilia following perforating injury. Traumatic cataract. Diffuse illumination.

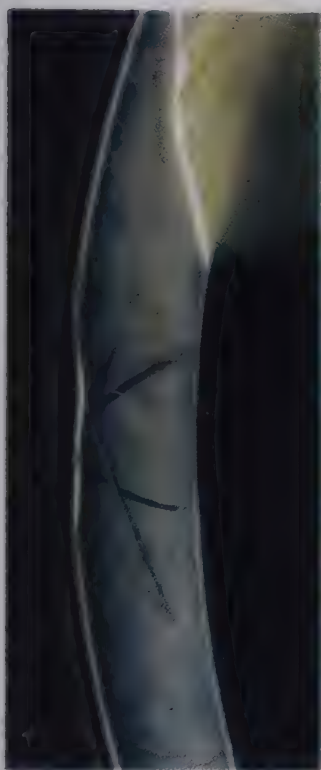
FIG. 6. High power (direct focal illumination) showing scar traversing the entire corneal thickness. Enlarged view of implanted hair follicles. Partially separated posterior synechiae with elongated patch of pigment on the anterior capsule of cataractous lens.



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prolapses following severe trauma are usually accompanied by definite dislocation of the lens. Small herniations of vitreous in the pupillary zone push the iris forward. Prolapse is most frequently seen after surgical procedures for cataract and is always found after discission of secondary membranes (after-cataracts).

Herniations of the vitreous into the anterior chamber can be easily overlooked, especially when the vitreous retains its transparent structure without alteration. In order to outline its surface a narrow beam, a high intensity of light, and moderate magnification are valuable. The eyes of the observer should be dark-adapted. Lateral oscillation of the beam is helpful. Prolapses may be large or small and in some cases may be seen to fill the entire anterior chamber.

Following contusions (nonperforating injuries or after intracapsular cataract extraction) the narrow beam will outline the prolapsed surface of the vitreous as a delicate gray line more or less regular in contour. The presence of pigment or blood on the surface or just behind the surface will help to delineate the prolapse. Because variations exist in the relucency and structure of the vitreous (normal or abnormal) the biomicroscopist is aided in the identification or diagnosis of the relucient structures within its substance. This is particularly true after discission when the changes produced by the perforation into the vitreous itself cause the prolapsed mass to appear more opaque and fluffy.

Blood corpuscles may be found within the prolapsed vitreous masses or outlining the surface of the herniation. An old hemorrhage may gravitate to the lower portions of the herniated mass and present a horizontal upper border (similar to a hyphema). Such a condition may remain for weeks. The outer membrane on the surface of the herniated vitreous is not permeable to blood corpuscles. As a result of condensation of the herniated vitreous surface, a thicker membrane is formed. Further condensation of the vitreous is seen as a webbing of light white threads which float with the movements of the eye.

In most cases, pigment granules may be observed on the surface or more commonly within the vitreous substance itself, arranged in lines or attached to threads.

Vitreous prolapse can be differentiated from fibrin networks by the fact that it usually shows numerous pigment granules, a more definitive surface, and because it seems to spill over the pupillary margin. The surface edge of large prolapses sometimes presents a scalloped appearance.

FOREIGN BODIES IN THE ANTERIOR CHAMBER

The literature contains reports of foreign bodies in the anterior chamber, comprising practically every substance known to mankind. Traumatic or surgical opening of the anterior chamber accounted for the presence of most of these substances. Except for certain metallic bodies, such as iron or copper, the reaction to most foreign substances is so minimal that they are often overlooked. Frequently, when they are small, only careful biomicroscopic or gonioscopic studies reveal their presence.

After surgical intervention or corneal perforation it may be discovered that cilia have become implanted in the anterior chamber by accident. These are usually attached to the iris. With the biomicroscope the cilia are seen to be covered by a delicate translucent film resulting from encapsulation by fibrinous exudate and a foreign body reaction (Plate XXXIX, figs. 5, 6). Cilia seem to be well tolerated in the anterior chamber. I have seen numerous cases in which, following cataract surgery, wisps of cotton were attached to the iris, probably from improperly prepared cotton tampons used for sponging. Likewise, small particles of rubber have been seen in the anterior chamber as a result of the washing-in of detached pieces from an irrigator, or from medicine dropper bulbs. In another case, a small fragment of a black silk suture was deposited on the anterior surface of the iris.

All manner of foreign bodies may be introduced into the anterior chamber following perforating injuries. The most common are fragments of metals, glass, stones, enamels, wood and cilia, and

carbon and gunpowder particles following explosions (Fig. 262).

There have been many reports of penetration of caterpillar hairs into the anterior chamber. The migration of the toxic barbed hair



FIG. 262. Perforating scar in cornea caused by piece of shattered spectacle glass; part of glass is seen in the anterior chamber.

may result in violent iridocyclitis and nodular iritis. Biomicroscopically, these hairs may be seen in the anterior chamber. Vogt described a case in which two hairs were found in the anterior chamber four months after their entrance. They were situated in the periphery and were spear-shaped, yellowish brown in color and surrounded by a grayish envelope (evidently a foreign body reaction). In the overlying cornea there were many deep vessels.

INFLAMMATORY CHANGES OF THE ANTERIOR CHAMBER

Inflammatory changes of the anterior chamber are of two types: those that occur in the boundary walls of the chamber and those that occur in its contents. The pathologic alterations of the borders (posterior corneal surface, the anterior iris surface, anterior lens

capsule) are discussed under the tissue of which each is a part. The alterations to which the contents of the anterior chamber (aqueous) are subject will be treated here.

CHANGES IN THE AQUEOUS

The importance of this subject in the problems of differential diagnosis cannot be overemphasized. This is especially true in differentiating early iritis, or the onset of irritation of sympathetic ophthalmia (in which the presence of cells or aqueous turbidity indicates intra-ocular disease) from conjunctivitis. The visible pathologic changes in the aqueous caused by inflammation are due to the presence of exudates derived from the surrounding tissues. The presence of exudates in the aqueous is always characterized by an *aqueous flare* or increased Tyndall phenomenon attributable to increase of protein content.

Varieties of Turbidity in the Aqueous. Diffuse turbidity of the aqueous without corpuscular elements. In iridocyclitis marked increase in the aqueous flare frequently occurs without the presence of cellular elements visible even with highest magnification or strongest illumination. In such cases, a simple increase of protein content of the aqueous is responsible for the presence of the flare. Quantitative mensuration of this Tyndall effect (tyndallimetry) can be performed with the Roenne colloidometer (page 560).

Turbidity due to the presence of corpuscular elements. Although in most cases simple turbidity of the aqueous is accompanied by varying proportions of suspended cells, which reveal the thermal convection currents, it is possible for the preponderance of cells to be so great as to cause marked diminution of aqueous transparency (trauma, hemorrhage, iridocyclitis and sympathetic ophthalmia). The corpuscular elements found in the aqueous may include all the cellular elements of the blood, degenerative products, and, in addition, pigment cells or granules coming from the uveal tract. Any or all of these elements may be precipitated on the posterior corneal surface. The tendency to precipitation depends directly on the thermal convection currents, the weight of the cells (gravity),

agglutination potential of the cell (surface tension), and the condition of the endothelium. The erythrocytes and polymorphonuclear leukocytes have less tendency than the lymphocytic cells to adhere to the cornea. The large mononuclear phagocytes have the greatest tendency to adhere to the cornea and to agglutinate with themselves. Most of the clear cells on the back of the cornea are usually lymphocytic or mononuclear unless the erythrocytes and polymorphonuclear leukocytes are present in great amounts. This fact has been proved histologically since such fine differentiation is not possible with the biomicroscope. *En masse*, fresh erythrocytes (e.g., as in hyphema) appear bright red in color, but when suspended in the aqueous and viewed with transmitted light they have a yellowish color. Blood staining of the cornea, however, which occurs after long-standing hyphema, is due to penetration of hemoglobin degenerative products (hematin) through a damaged or altered endothelial-Descemet's membrane barrier. Such staining gives a greenish gray discoloration to the corneal stroma. Uveal pigment, whether present singly or in clumps, appears dark brown in color. Leukocytes, on the other hand, when present in masses such as those seen in hypopyon appear as a whitish or yellowish white deposit. Above the level of the hypopyon the dense suspension of leukocytes causes a marked turbidity of the aqueous, appearing as whitish dots.

Formed opacities in the aqueous. At times coagulation of fibrin about suspended cellular elements may cause enlarged discoid aqueous opacities. Whitish fibrin coagulations in the aqueous may result in filiform, reticular or cobweb opacities, which may or may not be directly connected with the posterior corneal surface (Plate XL, fig. 2). On occasion, a pseudomembrane may be seen (Plate XL, figs. 3, 4). Such membranes may lose their early attachment to the iris. In interstitial keratitis as well as in other forms of chronic kerato-iritis, fibrinous filaments may become converted into glass-like hyaline striae. This has been attributed to covering over of the fibrinous strands by proliferated endothelial cells. As a rule reticular formations, whether fibrinous or hyaline, become dusted with pigmentary deposits.

PLATE XL

FIG. 1. A perforating injury of the cornea and lens. Scars through the corneal thickness. Lens matter in the anterior chamber.

FIG. 2. Filiform exudates on the posterior corneal surface extending into anterior chamber in plastic iritis. Aqueous flare in pupillary area showing cells and exudates.

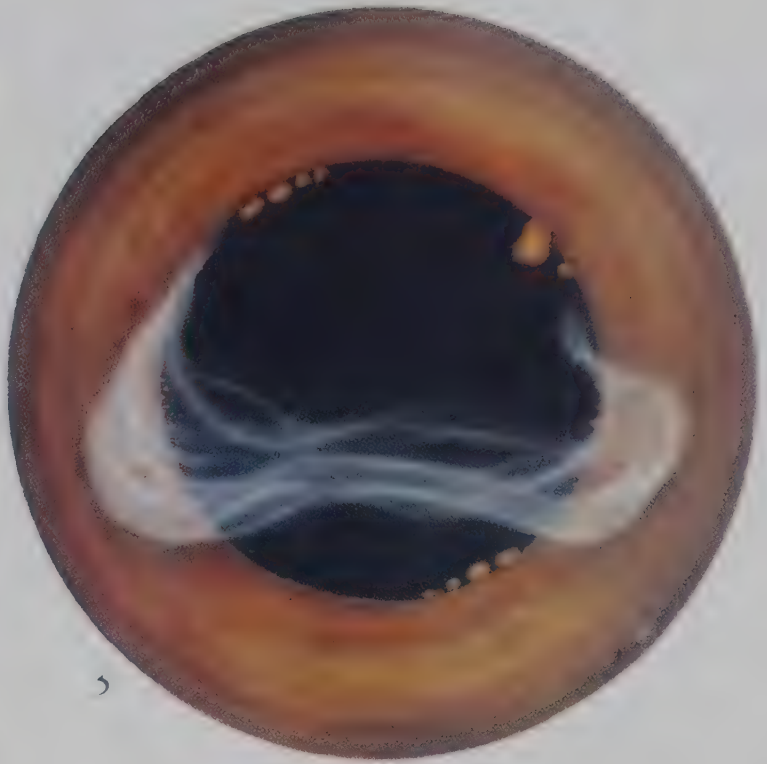
FIG. 3. A residual veil (diffuse illumination), floating like a sail, in the anterior chamber with both ends attached to the pupillary border in a case of quiescent chronic iritis.

FIG. 4. Veil (seen in Fig. 3) in anterior chamber by direct focal illumination (optic section).

FIG. 5. Heavy exudation in the anterior chamber in interstitial keratitis.

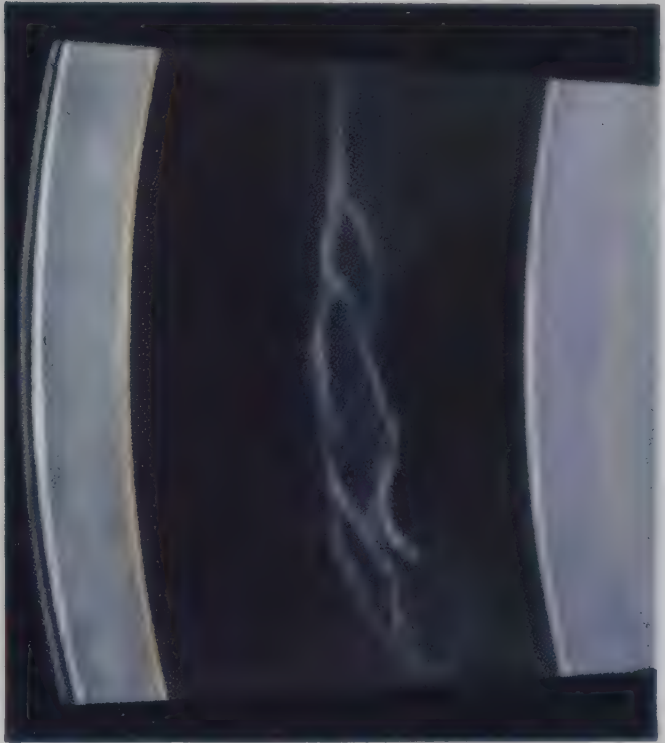


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In addition to hyphema and hypopyon formation, massive gelatinous collections may be seen, when the exudate is rich in protein (Plate XL, fig. 5). This condition may develop rapidly and the gelatinous mass may be so dense at the onset of iritis (especially in gonococcal iritis) as to obscure the iris. During the gel-like phase no thermal convection currents are discernible. These dense gelatinous exudates may disappear entirely within a day or two, leaving only a marked aqueous flare with considerable cellular suspension which will then reveal convection currents.

NEOPLASTIC FORMATIONS

Metastases of neoplastic growths are usually found near the angle of the anterior chamber and in many cases may require gonioscopic examination. In most instances, neoplasms of the iris are susceptible of close examination with the biomicroscope. When enlarged and in contact with the posterior corneal surface, they may block the entrance to the angle. Metastatic cells from an intra-ocular glioma or melanosarcoma may be carried by the intra-ocular current into the anterior chamber and may be deposited in its peripheral portions. Unless irritation is present the aqueous itself may be clear.

CRYSTALLINE DEPOSITS IN THE ANTERIOR CHAMBER

Many authors have reported instances of crystals (most of which resemble cholesterol) in the anterior chamber. They were found chiefly following injuries, hemorrhages, and in phthisical globes. Rarely, in certain disturbances of xanthine metabolism (gout, uremia), urate crystals may be seen in association with local intra-ocular inflammation.

Cholesterol appears as highly iridescent colorful needle-like or rectilinear crystals, either freely suspended in the lower parts of the anterior chamber or attached to the posterior corneal surface or iris. In some instances, they may fill the entire anterior chamber. In such a case described by Hughes,¹⁵⁵ secondary glaucoma developed. The crystals which were removed by paracentesis were shown to

be composed of cholesterol; reduction of tension followed their removal.

In other instances cholesterol may form a tumor-like mass of crystals in the angle enclosed by proliferated endothelium. In a case described by Koby (seen fifteen years following a perforating injury by a particle of iron), the crystals were enclosed in a gelatinous mass, which was shown to be vitreous. Histologic examination revealed that the crystals were cholesterol. In this case the crystals, which had been well tolerated in the vitreous and posterior chamber, caused an inflammatory reaction when they appeared in the anterior chamber. The lower and deeper parts of the cornea became vascularized.

Under the name of *scintillatio nivea*, whitish globules, resembling those seen in asteroid hyalitis, have been described in the anterior chamber. In the cases reported* this condition seemed to be associated with a prolapse of vitreous into the anterior chamber in aphakia or following luxation of the lens.

In this connection the difference between *synchysis scintillans* and asteroid hyalitis must be kept in mind in order to avoid confusion when describing them. In the latter, which occurs chiefly in the vitreous of the aged, in apparently otherwise normal eyes, showers of gleaming golden bodies are seen by ophthalmoscopic examination, while in direct focal illumination the biomicroscope reveals them as "dead-white" gleaming spherules of varying sizes. Chemical analysis has shown that they consist of calcium soaps of the fatty acids. On the other hand, *synchysis scintillans* usually follows ocular disease associated with fluid vitreous; it is frequently bilateral and may occur at any age. In this condition the bodies are more crystalline in appearance (needle-like), having a glittering golden or silvery, tinsel-like character, even in direct focal illumination. Furthermore, their movements are unrestrained and they may settle to the bottom of the vitreous, in contrast to the asteroid bodies, which move in a wavelike fashion and return to their original sites. Chemically, the crystals of *synchysis scintillans* have been shown to be composed of cholesterol.

* Evans' and Lloyd's cases.

PATHOLOGIC VARIATIONS IN THE DEPTH OF THE ANTERIOR CHAMBER *

In addition to variations in depth of the anterior chamber caused by refractive errors and congenital anomalies, pathologic states may alter the depth of the anterior chamber.† Even when the anterior chamber has been apparently abolished, immediately after perforating injury or surgery, the narrow beam will reveal some residuum of the anterior chamber. Naturally, this will always be found in the pupillary zone, since the thickness of the iris prevents the anterior lens capsule from coming into contact with the posterior corneal surface. Vogt has shown that similar areas of residual chamber may be discovered even when the anterior chamber appears to be completely effaced in other areas distant from the pupil.

The classic picture of shallow anterior chamber is found in acute glaucoma. Narrowing of the anterior chamber may occur following the formation of adherent leukomas or other anterior iridic synechia in trauma or disease. In *seclusio et oclusio pupillae* blocking of the pupil gradually causes a deepening of the posterior chamber with a consequent diminution in depth of the anterior chamber owing to the arching forward of the iris between the fixed extremities.

The extreme picture of this is seen in iris bombé. The evaluation of the depth of the anterior chamber is of great importance in all surgical procedures involving the anterior segment.

On the other hand, increases in depth due to pathologic states frequently occur. This is seen in hypotensive states following trauma with or without retinal separation, luxation of the lens, or fluid vitreous.

Ectatic conditions of the cornea following corneal disease (e.g., interstitial keratitis, keratoconus, or thinning of a scarred cornea) may lead to increase in depth of the anterior chamber.

* See also Congenital Alterations of the Anterior Chamber, page 568.

† See pages 559, 608 for methods of measurement of the depth of the anterior chamber.

EXAMINATION OF THE CHAMBER ANGLE WITH THE BIOMICROSCOPE *

The procedure for examining the angle of the anterior chamber with the biomicroscope was developed by Koepe. Since Tron-

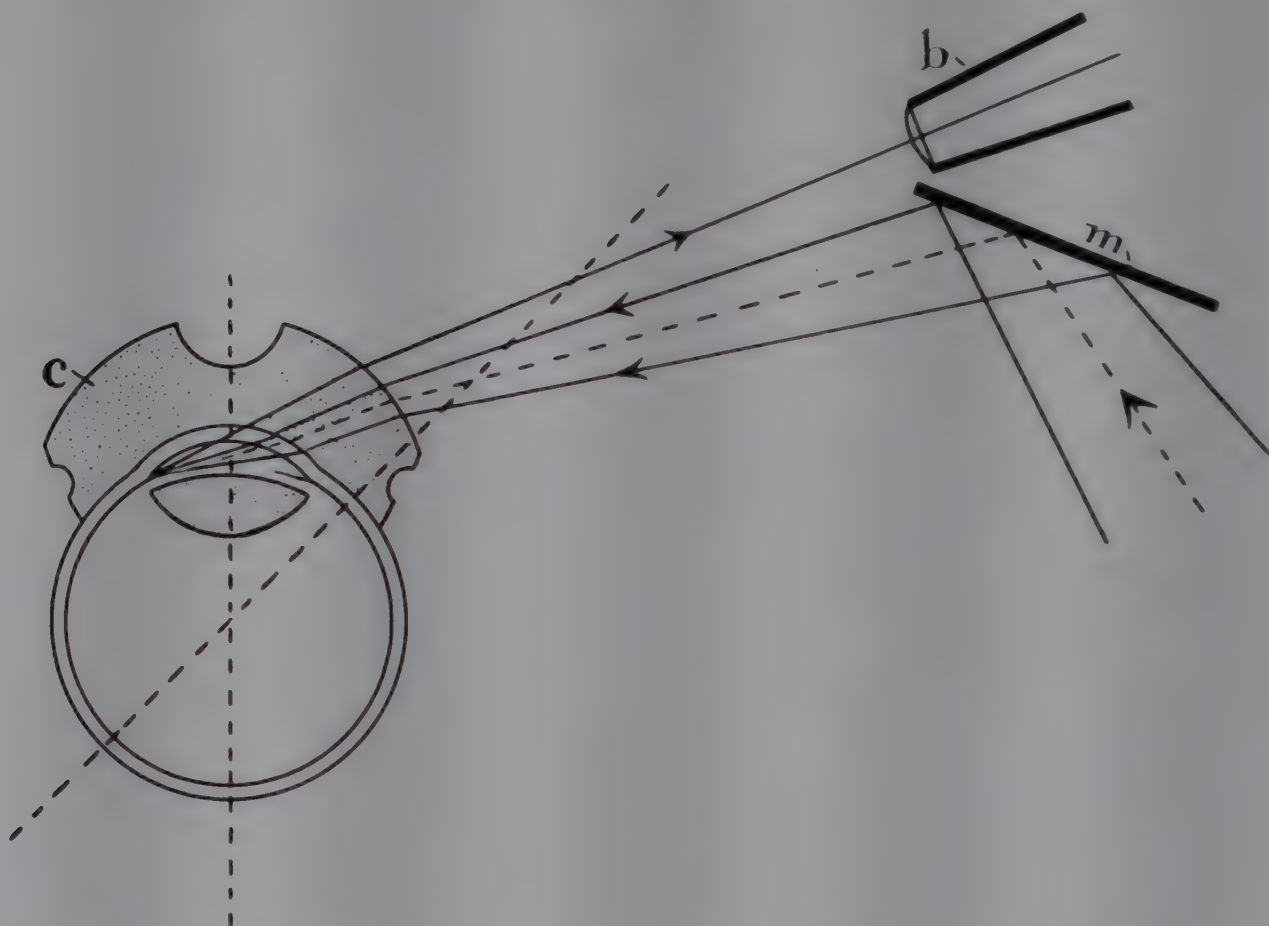


FIG. 263. Diagram showing the direction of the observation and beams through the Koepe contact glass (*c*) for examination of the angle of the anterior chamber. The Kleefeld silvered mirror (*m*) changes the direction of the rays so that they almost correspond to the line of observation. *b*. Monobjective stereoscopic microscope.

coso simplified the technique by using the gonioscope, biomicroscopy for this purpose has become less common. Biomicroscopic examination of the angle of the chamber, according to Koepe's technique, had many practical disadvantages. First, it was impossible to examine the entire angle circumference with the patient in the sitting position. Second, Koepe's contact glass (with the patient in the sitting position) required some contrivance for its retention. Finally expensive accessories, such as the monobjective microscope and silver mirror, were needed.

In order to inspect the angle of the anterior chamber a contact

* See Chapter 17.

glass is required to overcome the curved refractive corneal surface and to prevent total reflection of tangential rays within the cornea. When the illuminating beam is passed almost tangentially through the cornea toward the angle across the chamber the rays are reflected superficially or within the cornea (scatter) with the result that very little of the incident light reaches the angle. Another difficulty lies in the fact that the emergent beam is reflected back to the periphery of the opposite iris or angle because of internal reflection. By the prismatic action of the contact glass the exit beams from the angle are refracted so that they can emerge and be seen with the microscope (Fig. 263).

Historically, Trantas was the first to inspect the angle of the anterior chamber of the living eye. He made his observations with the ophthalmoscope while exerting pressure on the opposite side of the globe. Salzman was the first to employ a contact lens. He used a Ficke contact glass and later a magnifying contact glass, both of which proved somewhat unsatisfactory owing to distortion. Later, Koeppe designed a special contact glass for chamber examinations with the biomicroscope. This contact glass had a depression in its anterior surface so that a knot in a bandage could serve to retain it.

KOEPPE'S METHOD

The cornea is anesthetized, miosis obtained whenever possible with eserine (physostigmine) and the contact glass (with saline solution) inserted between the eyelids and secured with a knotted bandage or other device. The patient is then seated at the instrument. A silvered mirror is attached to the illuminating lens at a 45 degree angle in order to direct the beam tangentially across the anterior chamber into the opposite angle. The axis of observation is as close to the beam as feasible. A 100 mm. illuminating lens is preferred because the working distance is greater than with a 70 mm. lens.

Chapter Seventeen

GONIOSCOPY

By H. SAUL SUGAR

ALTHOUGH introduced into ophthalmology more than three decades ago, gonioscopy has not been given more than a few paragraphs in any ophthalmic textbook* and only recently has passed beyond its adolescent phase. The term "gonioscopy" was introduced by Trantas in 1907 and, independently, by Troncoso in 1921, to designate that portion of biomicroscopy which deals with observation of the angle of the anterior chamber of the eye.

The first chamber angle observation in a living subject was made by Trantas. Using direct and indirect ophthalmoscopy, he succeeded in seeing the nasal and temporal portions of the angle in approximately one quarter of his cases. When the chamber was too shallow to make observation possible, he found that digital pressure on the globe through the eyelid brought it within range of vision.

Mizuo, in 1914, brought out the fact that the lower portion of the chamber angle could be made visible by instilling water into the lower conjunctival cul-de-sac after pulling the lower eyelid away from the globe. In the same year Salzmann published an excellent description of the angle as seen ophthalmoscopically. Originally he used Ficke's keratoconus contact glass to observe the angle; later he employed an improved contact lens of his own design combined with instillations of saline solution between the cornea and the lens. Since his time all investigators have used contact lenses

*Since the writing and revision of this material, two books on gonioscopy have appeared: (1) TRONCOSO, M. U.: *A Treatise on Gonioscopy*. Philadelphia, Davis, 1947; (2) BUSACCA, A.: *Elements de gonioscopie normale, pathologique, et experimentale*. Sao Paulo, Brazil, Tipografia Rossolillo, 1945.

of various types for observation of the angle of the anterior chamber. Koeppe devised a lens suitable for slit lamp technique. A special mirror permitted observation of the temporal and nasal portions of the angle. Lenses of the original Koeppe design are still used today (the Koeppe "A" lens).

The era of modern gonioscopy began in 1925, with a paper by Troncoso in which he described the gonioscope, a monocular hand microscope with a nearly coaxial source of illumination, and a new contact lens which he had suggested to Koeppe (the Koeppe "C" lens). He pointed out that synechias are the result and not the cause of glaucoma. (Cases of glaucoma following cataract operations were not included in his studies.) With Castroviejo he contributed to our knowledge of the comparative anatomy of the angle of the anterior chamber. His subsequent papers established his place as a pioneer and authority in this field.

Thorburn, in 1927, following a study of 100 cases of both the narrow angle and simple glaucoma which he examined with a loupe and flashlight, agreed with Troncoso that synechias are a result rather than a cause of these types of glaucoma. Werner, in 1932, gave the first description of the appearance of the angle following iridencleisis operations, and came to the same conclusion.

When Barkan, in 1936, introduced his operation of goniotrabeculotomy, the need for better knowledge of the anatomy of the angle brought about a surge of general interest in gonioscopy.

GONIOSCOPIC TECHNIQUE AND APPARATUS

The recesses of the angle of the anterior chamber are not normally accessible to either direct or slit lamp observation because of the curved refractive corneal surface and total reflection of tangential light rays. With the use of a diagnostic contact lens, however, the emerging rays are so refracted as to render the angle easily visible. The contact lens not only acts as a prism which transmits the emergent rays from the angle region to the observer, but magnifies the area significantly. It is thus the basic instrument in gonioscopy.

Various adjuncts such as a source of illumination, a suitable fluid between the cornea and the gonioscopic lens to produce optical continuity, and magnifying instruments, including the binocular loupe,

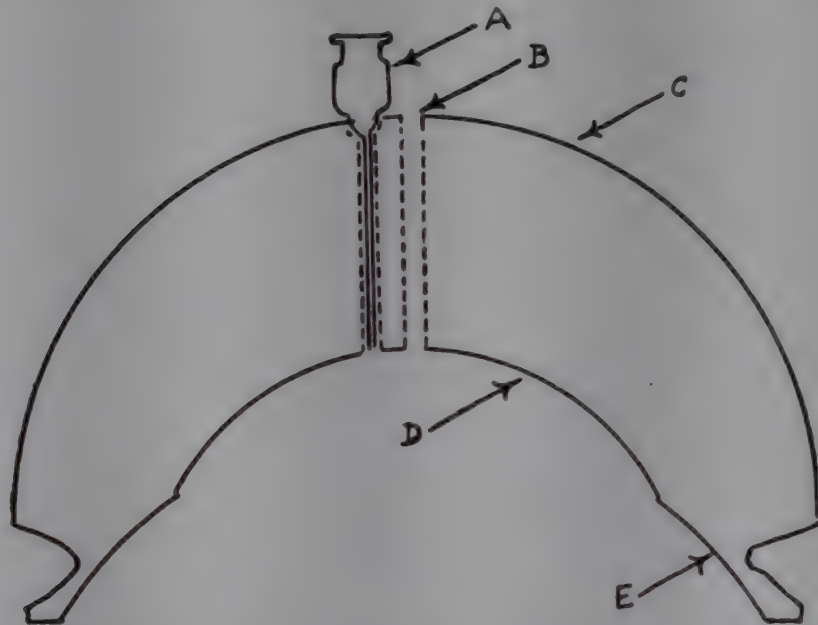


FIG. 264. Friedman contact lens with channels for introducing fluid and permitting egress of air. A, Blunt hypodermic needle for introducing fluid; B, air channel; C, surface of contact lens; D, under surface of contact lens; E, scleral shoulder. (Courtesy of Obrig Laboratories, Inc.)

the corneal microscope, or other magnifiers such as Troncoso's various gonioscopes, serve to complete the equipment for observation of the structural details of the angle.

Gonioscopic contact lenses are made of ophthalmic glass or of one of the new plastic materials, such as lucite. Those made of ophthalmic glass are most satisfactory since their optical surfaces are more perfect and do not scratch easily. They are, however, considerably more expensive and are breakable. The surfaces of the plastic lenses scratch rather easily and do not "wet" as well as do those made of glass. It is for this reason that air bubbles frequently remain under the plastic lenses after the fluid is instilled. Friedman has attempted to overcome this disadvantage by having two narrow channels drilled through the lens — one at the center, and the other 2 mm. away — and filling the space between the lens and cornea through the more eccentrically placed channel with a short blunt-pointed needle. The air escapes through the central hole (Fig. 264).

Three types of diagnostic lenses are available for ordinary gonioscopy. The first two were designed by Koeppe. They are the "A"

lens and the more convex "C" lens suggested by Troncoso (Fig. 265). The original "A" lens has a central depression in its surface which was used to hold the lens in place. A lens designated as a "B" lens

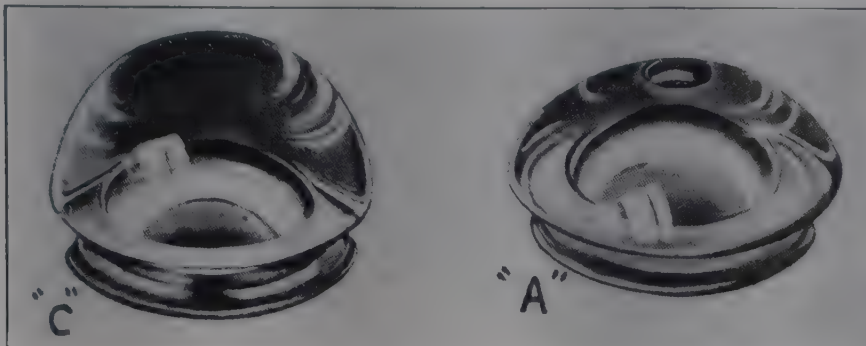


FIG. 265. Contact lenses used in gonioscopic examination. (After Troncoso.)

(Fig. 266) is not used in gonioscopy but for slit lamp examination of the vitreous.

The "C" lens (Fig. 267) has the same inner dimensions as the "A" lens. Its inner surface is concave and is shaped to conform to the curvatures of the cornea and adjacent sclera. The outer surface

BAUSCH & LOMB CONTACT GLASS B



FIG. 266

FIG. 266. Bausch & Lomb contact glass B. (Courtesy of Bausch & Lomb Optical Company.)

BAUSCH & LOMB CONTACT GLASS C

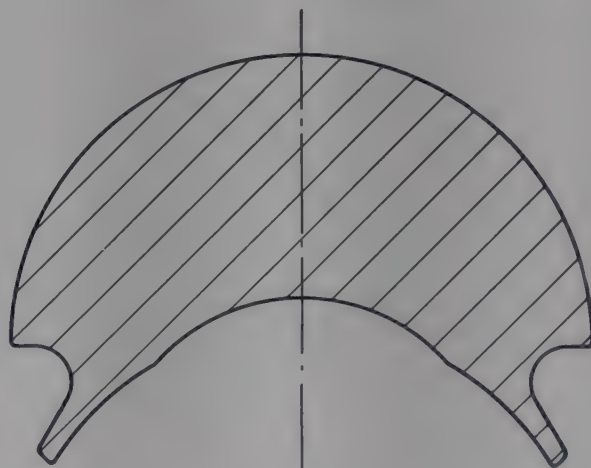


FIG. 267

FIG. 267. Bausch & Lomb contact glass C. (Courtesy of Bausch & Lomb Optical Company.)

is more convex than that of the "A" lens, but like the latter, it has a circular groove around its circumference into which the eyelid borders fit and thus keep the lens in position (Fig. 268). The "C" glass produces more prismatic distortion than the "A" glass but gives higher magnification. It is better for goniophotography due to the absence of any depression in its surface. A plastic "A" lens

with a detachable handle has been devised by Barkan (Fig. 269). The "C" glass is also available in plastic.

The third type of gonioscopic lens for ordinary gonioscopy is the

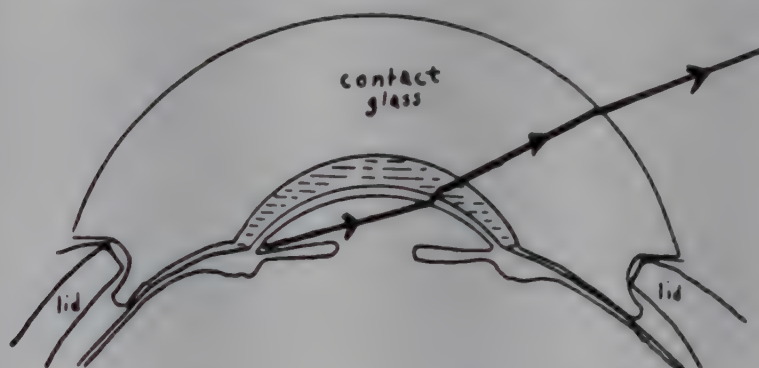


FIG. 268. Schematic diagram showing contact lens held in place by eyelids. The arrows show path of emergent ray from the angle.

"Goniolens" devised by Troncoso (Fig. 270). This lens is particularly useful in instances where the palpebral fissure is too small to permit

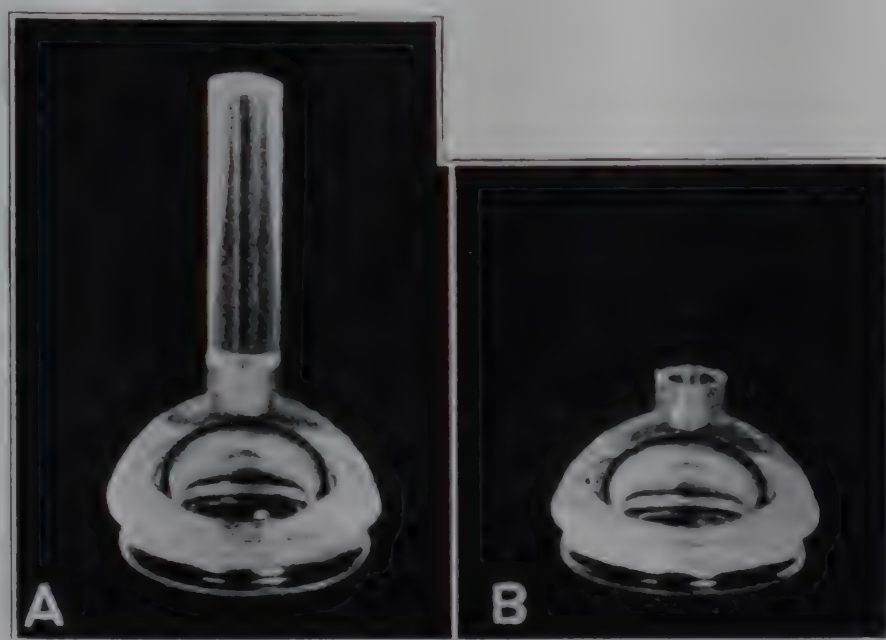


FIG. 269. Barkan's plastic contact lens. A. With handle attached. B. Without the handle. (Otto Barkan.)

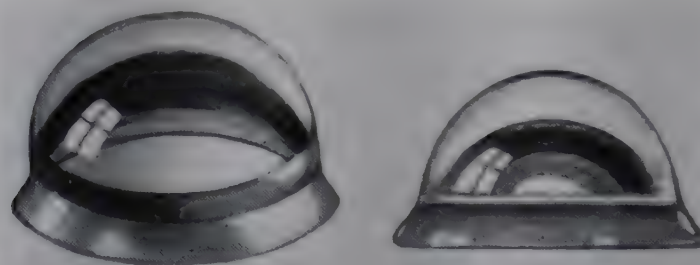


FIG. 270. Troncoso's new "Goniolens." (Courtesy of Dr. M. U. Troncoso.)

insertion of the other lenses. There is a tendency for the lids to slip over its surface. Because of some difficulty, at times, in removing

the lens, I had a detachable handle inserted into its apex to overcome this disadvantage. However, the lenses now are made with a notch in the rim to permit insertion of a lacrimal needle, through

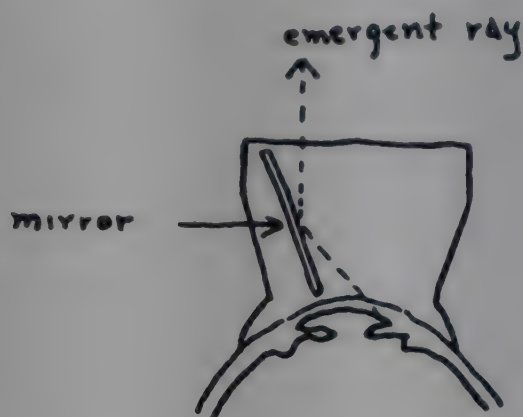


FIG. 271. Goldmann's contact lens for use with the slit lamp. (Goldmann.)

which the solution is injected and which may be helpful in removing the lens.

For slit lamp gonioscopy, two plastic lenses are available. The first is that devised by Goldmann. This is a flat-surfaced lens (Fig. 271) which contains a mirror placed at an angle of 64 degrees with the flat surface. The lens is rotated to view the different meridia of the angle circumference while the patient is seated in front of the biomicroscope. A special prism attached to the slit lamp permits the observation-illumination angle to be reduced to 5 degrees.

The second lens for slit lamp gonioscopy was devised by Lee Allen. This lens is a prism lens segment with one surface which fits the cornea directly. A second surface acts as a reflector. This ingenious device can be rotated in a frame so that all portions of the angle circumference can be observed (Figs. 272, 273).*

Choice of a proper fluid to fill the space between the contact lens and the cornea depends on the time required for examination. Prolonged use of physiologic saline solution and water leads to epithelial edema which obscures details of the image. A sodium carbonate buffer solution (Gifford) with a pH of about 7.3 is

* Mr. Allen, in a personal communication, has compared his lens with Goldmann's as follows: "I have great admiration for Dr. Goldmann's contribution of the mirror principle. On the other hand, the posterior portion of his glass (that part which contacts the eye and lids) is fundamentally the same as in all preceding lenses. My attempts at improvement have been directed first toward better maintenance of optical continuity between the contact glass and cornea, and secondly toward more convenience in its use with standard slit lamp biomicroscopes."

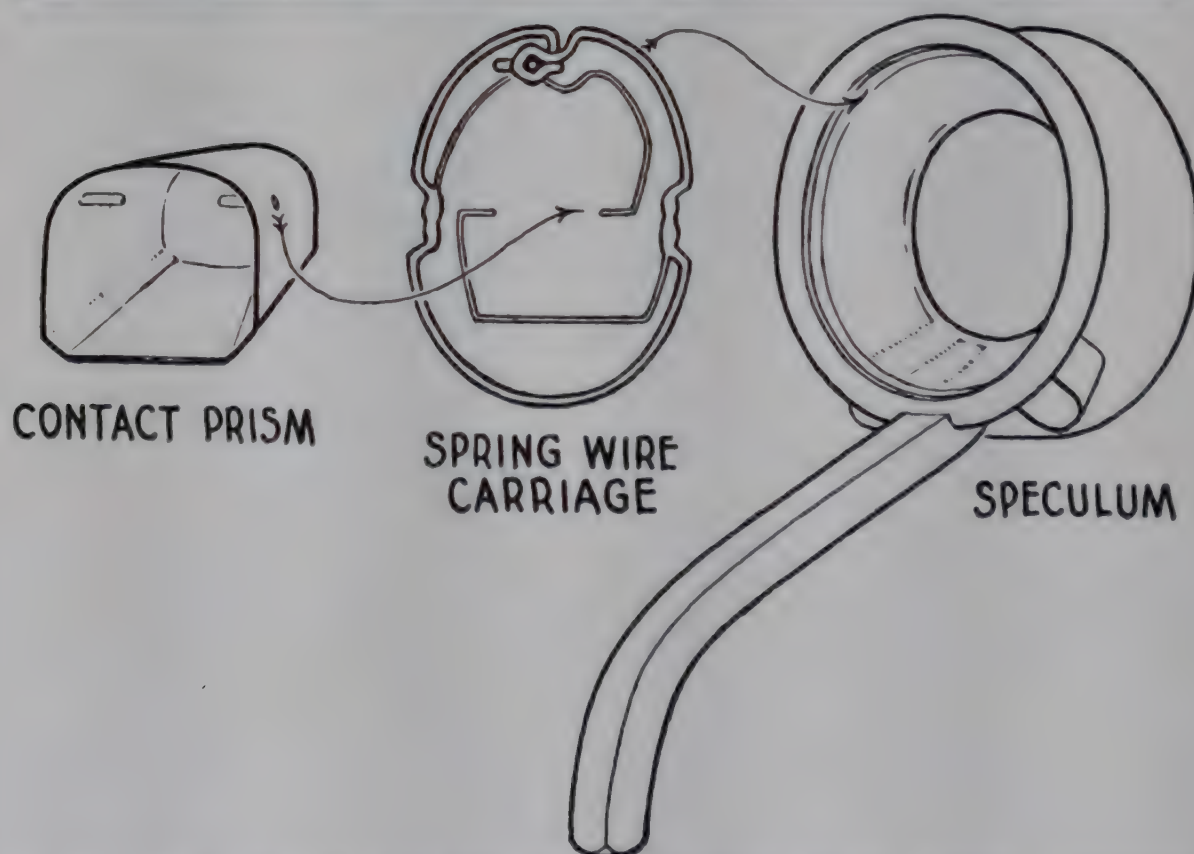


FIG. 272. *Top:* The Lee Allen prism gonioscope in place on the eye. *Bottom:* Component parts of the prism gonioscope.

usually satisfactory.* Cogan suggests a hypertonic (1.5 per cent) saline solution for this purpose. Swan has advocated the use of 1 per cent methyl cellulose solution.

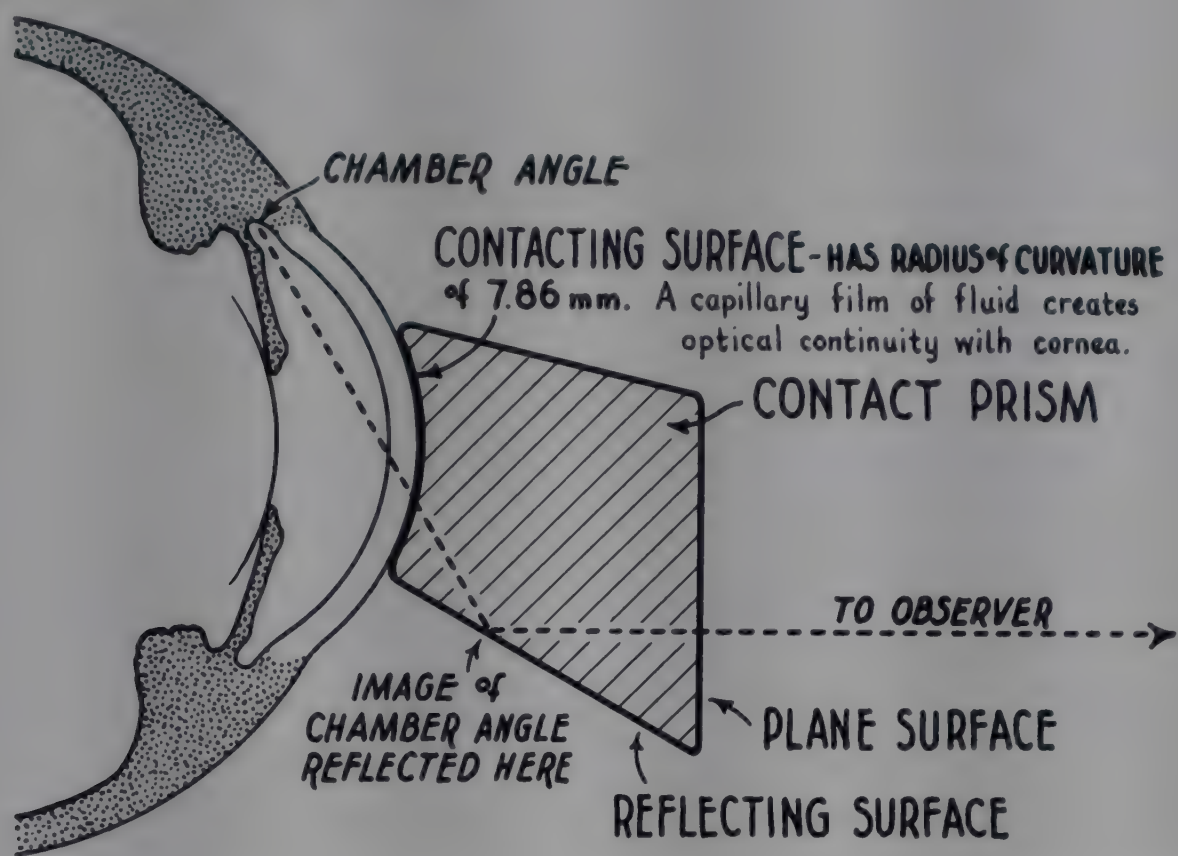


FIG. 273. Illumination of the anterior chamber during examination with the prism gonioscope.

For use with any of the plastic lenses, one of the wetting agents such as zephiran or phemerol in aqueous solution is particularly useful to keep the lens in before use. This not only aids materially in avoiding bubbles but is excellent for antisepsis.

The gonioscopic contact lens is inserted while the patient is recumbent. Anesthesia of the cornea and conjunctiva is induced with a drop of 0.5 per cent pontocaine hydrochloride solution, repeated at an interval of two or three minutes. The lower lip of the contact lens is inserted under the edge of the lower eyelid and the patient is asked to look toward his feet. The upper eyelid is pulled over

* This solution is made up as follows, according to the formula of Dr. Sanford R. Gifford:

Solution 1:	
Boric acid crystals	12.4 gm.
Reagent potassium chloride	7.4 gm.
Distilled water	1000 c.c.
Solution 2:	
Anhydrous reagent sodium carbonate	21.2 gm.
Distilled water	1000 c.c.

To each 30 c.c. of solution 1 add 1.125 c.c. of solution 2 to give a pH of 7.3.

the upper lip of the lens. The patient's head is then turned toward the opposite side and the temporal edge of the lens is lifted slightly. The solution used to fill the space between the cornea and contact lens is introduced with an eyedropper until no air bubbles remain beneath the lens. Momentary slight pressure is made on the lens to displace any excess fluid and to produce a slight negative pressure beneath the lens. The patient's head is then returned to its original position.

Examination of the lens-covered eye is made with a magnifying optical system. One may use the binocular loupe for examining aphakic eyes with their wide angles and for determining the gross condition of the angle after surgical procedures. For careful examination to determine the presence or absence of synechias especially in eyes with narrow angles, a microscope must be used. The binocular corneal microscope, with a magnification of from 10 to 14 diameters, is most satisfactory for this purpose. A monocular instrument such as the Troncoso gonioscope or his more recent monocular microscope may be used (Figs. 274, 275). The original gonioscope consists essentially of a revolvable compound microscope with its illuminating system, mounted on a handle to which electrical connections are attached. The instrument has two square prisms at the objective end of both microscope and illuminating system, arranged so that the illuminating and image-forming rays are coaxial. The original Troncoso gonioscope is no longer manufactured.

The microscope may be held in the braced hands or, preferably, be suspended from, or mounted on, a gonioscopic support. Barkan, Fine, Hartshorne, McAlester, McLean, and Bogart have published descriptions of their own instruments for this purpose. Gradle used a compact, balanced, wall bracket, such as is ordinarily employed to support portable x-ray apparatus.

In order properly to illuminate the field under examination, a focal illuminating system is necessary. A coaxial system, preferably attached directly to the microscope, is most satisfactory. The Barkan lamp affords an excellent source of illumination. For ordinary pur-

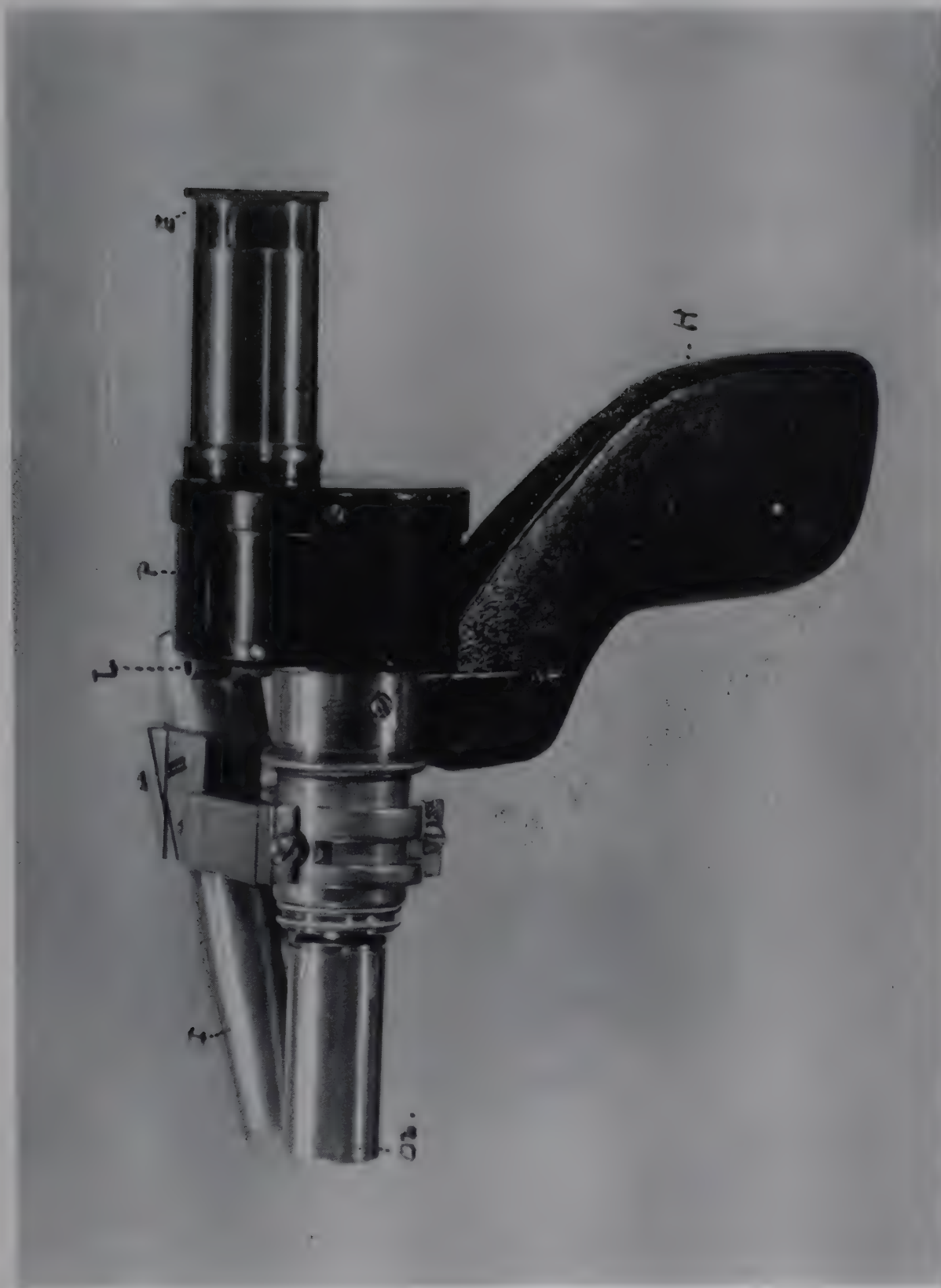


FIG. 274. Monocular gonioscope of Dr. M. Urive Troncoso. *Ob.*, Objective; *I*, illuminating system; *P*, redressing prism; *E*, eye piece; *L*, electric attachment to rheostat; *H*, handle. (Troncoso.)

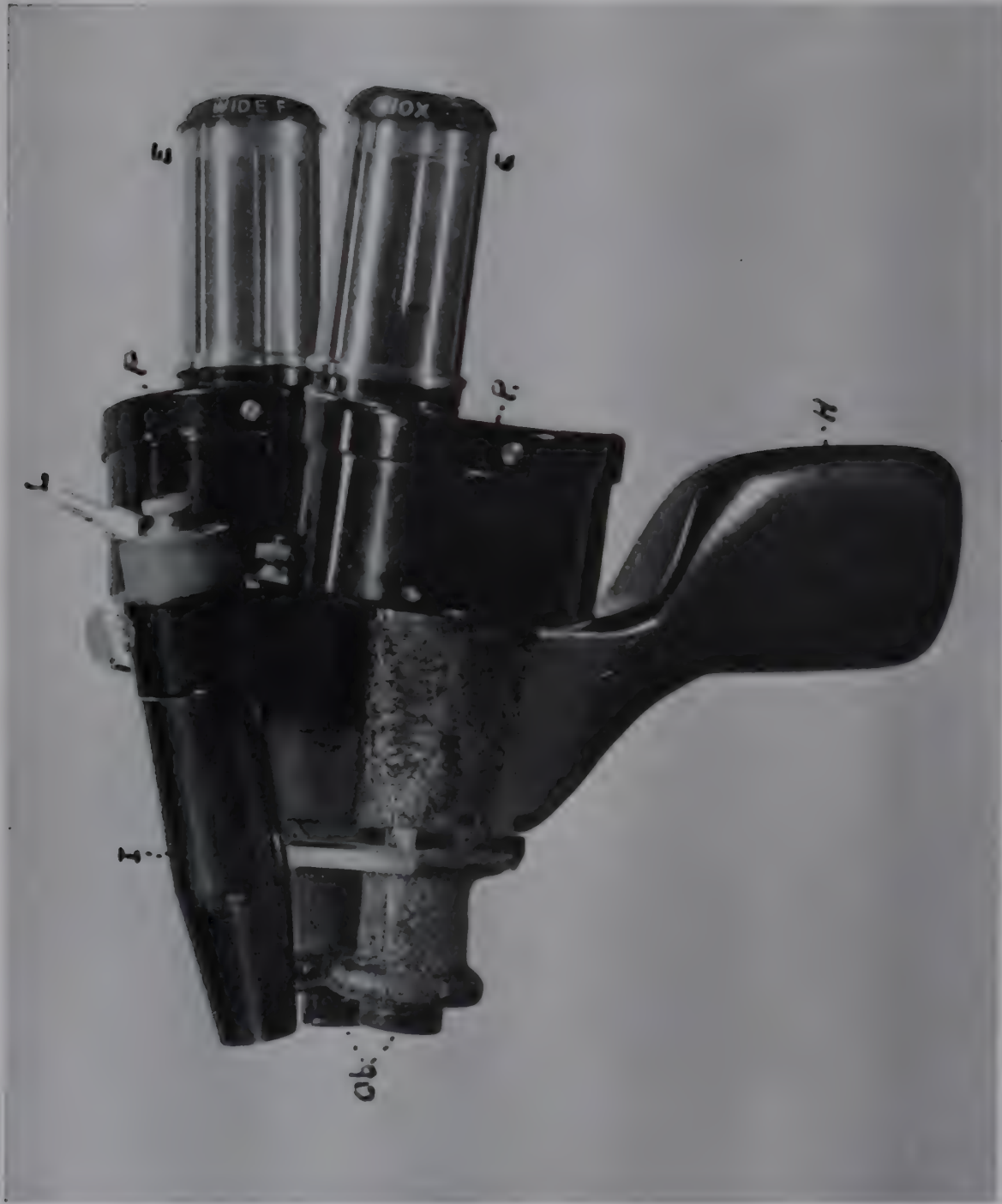


FIG. 275. Binocular model. *Ob*, objective; *I*, illuminating system; *P*, redressing prism; *E*, eye piece;
L, electric attachment to rheostat; *H*, handle. (Troncoso.)

poses, when using a binocular loupe, a small hand flashlight is all that is necessary.

Gonioscopic technique may be used in obtaining color photographs of the angle of the anterior chamber. I have made such photographs on type A Kodachrome film with a Kine-Exacta camera and an f2.8 Tessar lens mounted on a gonioscopic stand. A synchronized illumination system was used, consisting of a No. 1 photoflood lamp (nonfrosted) connected with a resistance to give about two footcandles of light. When the angle seen through the contact lens was properly focused, the synchronizing switch was pressed, shunting the resistance out of the circuit, and releasing the shutter. A momentary flash of about 175 footcandles was thus obtained. A standard exposure of 1/25 second at f2.8 was used. The photoflood lamp is housed in a hooded goose-necked desk lamp and is held 10 to 12 cm. from the contact lens.

GONIOMETRY

The physiologic importance of the angle of the anterior chamber, in glaucoma particularly, makes it necessary to study the measurement of the acuity of the chamber angle. Such a study may be termed goniometry.

Ordinarily one may assume that the angle acuity varies fairly constantly with the depth of the anterior chamber, but this is not always the case. Eyes with narrow angles and fairly normal anterior chamber depths are seen not infrequently. A method of measuring the angle acuity gonioscopically has been introduced by Gradle and Sugar. This method employs the Koeppe lens-microscope gonioscopic equipment with a micrometer graticule etched in tenths of a millimeter in one of the $5.5\times$ microscope oculars. Measurements are made simply by observing the length of an imaginary line between the apparent end of Descemet's membrane (Schwalbe's ring) and a point on the iris perpendicularly opposite it (Fig. 276). The angle of observation must be kept as nearly parallel to the plane of the iris as is possible. Since both walls of the

angle are considerably curved, it is impossible to measure the angle acuity with any great degree of accuracy; therefore for practical purposes the length of the line perpendicular to the iris at the end of

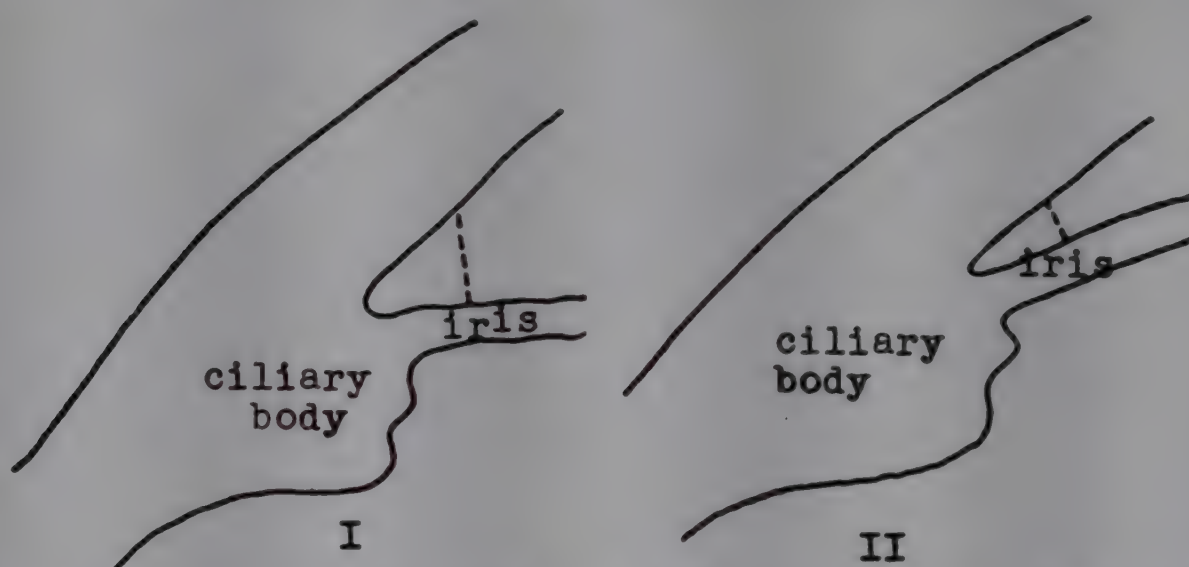


FIG. 276. Diagrammatic representation of the angle depth measurement in normal (I) and shallow (II) chambers. The broken lines are the perpendiculars to the iris at the termination of Descemet's membrane.

Descemet's membrane is the only measurement used. It is only approximate but is valuable for comparative measurements of different chamber angles and of the same angle at various times.

In order to get an approximation of the angle acuity in degrees one must first correct the value obtained for the perpendicular to the iris at the end of Descemet's membrane by using the graticule-microscope magnification factor and the magnification value for the contact lens. The corrected measurement may be considered the side opposite the angle being measured. The wall of the anterior angle may be assumed to be a straight line which measures approximately 0.8 mm. and becomes the hypotenuse of the right-angled triangle. The angle acuity is then derived by simple mathematical calculation. When the plane of the iris approaches the anterior wall of the angle the line perpendicular to the iris at the end of Descemet's membrane becomes shorter and shorter until, with extremely shallow chamber and angle depths, it finally becomes immeasurable.

Gonioscopic and goniometric observations indicate that the depth of the angle is the same in normal eyes and in eyes with glaucoma simplex. The eyes of individuals with acute glaucoma, on the other hand, have very narrow angles, as do the nonglaucomatous eyes

of patients with unilateral acute glaucoma. Apparently eyes with very shallow chambers are anatomically predisposed to acute glaucoma. Rosengren, in 1930, in a comprehensive study of the depth of the anterior chamber, came to the same conclusion.

A number of factors influence the depth of the anterior chamber. They may be divided into two groups: anatomic and physiologic. The anatomic group includes (1) errors of refraction, (2) continuous growth of the lens throughout life, and (3) size of the eye. The physiologic group includes (1) accommodation, (2) pupillary dilatation, and (3) congestion of the ciliary body.

ANATOMIC FACTORS AFFECTING THE DEPTH OF THE ANTERIOR CHAMBER

Errors of Refraction. It has long been known that acute glaucoma occurs almost always in eyes with shallow chambers and that the large majority of them are hyperopic. Fuchs stated that "a disposition toward inflammatory glaucoma [acute glaucoma with vascular congestion] appears to belong principally to hypermetropic eyes, whereas strongly myopic eyes are to be regarded as having almost complete immunity against the disease." Two facts indicate the truth of Fuchs' observation. First, gonioscopic observation indicates that in axially hyperopic eyes the root of the iris appears to be inserted farther forward on the anteromedial surface of the ciliary body, while in axial myopes it is inserted correspondingly farther back. Second, in individuals of the same age, an analysis of the anterior chamber depth and refractive error indicates that there is a rather definite inverse relationship between the two; that is, the chamber depth decreases as the hyperopia increases. This analysis is based on Rosengren's accurate measurements in 191 cases of hyperopia in a series of normal patients between the ages of 25 and 45 (Table VI), and on a second study of 398 normal patients at the Barnes General Hospital. The latter study also indicated that in eyes with the same refractive error, the chamber depth decreases with age.

TABLE VI

THE RELATION BETWEEN REFRACTIVE ERROR AND CHAMBER DEPTH

(The figures in parentheses indicate the number of cases in each group)

AGE	REFRACTIVE ERROR				
	0-0.50 D.	0.75-1.00 D.	1.25-1.50 D.	1.75-2.50 D.	2.75-3.50 D.
25	3.55 (3)	3.59 (8)	3.57 (5)		
26	3.79 (3)	3.39 (5)	3.36 (5)		
27	3.57 (4)				
28	3.38 (3)	3.48 (7)			
29	3.79 (2)	3.54 (3)	3.46 (3)	3.54 (2)	
30	3.80 (2)	3.63 (6)	3.12 (2)		
31	3.27 (6)	3.79 (6)	3.58 (2)		
32	3.43 (6)	3.36 (8)	3.16 (4)		
33	3.89 (2)	3.16 (2)	3.73 (2)		
34	4.15 (2)	3.71 (3)	3.22 (3)		
35	3.44 (7)	3.46 (5)	2.98 (3)	3.23 (1)	
36	3.85 (3)	3.60 (3)	3.38 (4)		
37	3.62 (2)	3.50 (2)		2.95 (2)	
38	3.44 (4)	3.61 (2)	3.20 (2)	3.51 (2)	
39	3.24 (4)	3.22 (7)	3.45 (2)		
40	3.42 (3)	3.32 (10)	3.07 (3)		
41	3.33 (7)	3.29 (6)	3.26 (3)		
42	3.49 (8)	3.48 (11)	3.50 (6)	2.83 (3)	
43	3.50 (6)	3.45 (7)	3.38 (10)	3.19 (3)	
44	3.38 (8)	3.44 (15)	3.32 (4)	3.09 (3)	2.91 (1)
45	3.45 (8)	3.37 (9)	3.26 (5)		
Average	3.56 mm.	3.46 mm.	3.33 mm.	3.19 mm.	2.91 mm.

In a third study of refractive error in 140 eyes with acute glaucoma from the practice of Drs. H. S. Gradle and S. J. Meyer, there was an average hyperopic error of 2.72 diopters. Only 7 of the 140 were myopic.

Continuous Growth of the Lens Throughout Life. As lens fibers are added to the lens cortex, the dimensions of the lens increase. This produces definite progressive shallowing of the chamber and the angle, as is shown in the following commonly accepted chamber-depth values:

AGE YEARS	PLANTENGA (1898) mm.	SAUNTE (1905) mm.	LINDSTEDT (1916) mm.	RAEDER (1922) mm.	ROSENGREN (1930) mm.
40-50	2.93		3.56	3.37	3.35
50-60		3.22	} 3.213	3.15	3.22
60-70	2.61			3.04	3.17

This shallowness is ordinarily of little significance except in persons in whom it reaches a marked degree. In persons with axial hyperopia in whom the depth of the chamber is relatively less than normal, it is probable that the increasing size of the lens is the chief factor in increasing the shallowness of the chamber.

Size of the Eye. Priestley Smith stated that the eyes of patients with glaucoma are on the whole a little smaller than normal. Although he did not distinguish between the various etiologic types of glaucoma, his studies indicate that mechanical factors associated with decreased ocular dimensions may be one of the factors in producing a relative disproportion between the increasing volume of the lens and the volume of the surrounding scleral envelope.

PHYSIOLOGIC FACTORS AFFECTING THE DEPTH OF THE ANTERIOR CHAMBER

Accommodation. In individuals of prepresbyopic age and to a much lesser extent in early cases of presbyopia, accommodation tends to decrease the angle width. This contradicts the previously held views of Grönholm and Thomson who concluded that accommodation opens the chamber angle. I observed, directly, the effect of accommodation on the angle by the following method: A gonioscopic contact lens was placed on one eye of a recumbent, practically emmetropic, prepresbyopic individual. He was asked to observe a printed card held about 12 inches from the other eye. A 4.00 diopter convex lens was placed in front of the reading eye and the angle of the lens-covered eye was observed with the binocular

microscope. When the reading lens was removed, a slight convergence movement was observed, followed, after a short period of lag, by forward movement of the ciliary zone of the iris. Observations on normal subjects indicated that the accommodative movement is strongest in young individuals, and decreases as age advances, becoming hardly perceptible in the late 40's and early 50's. The comparative amount of movement of the iris can be measured goniosmetrically.

Pupillary Dilatation. By increasing the thickness of the iris tissue in the angle, pupillary dilatation tends to decrease the angle but this decrease is significant usually only when the angle is relatively narrow. The relation between the chamber and angle, the size of the pupil and the thickness of the iris when the pupil is dilated are important in the mechanical blockage of the angle which leads to that type of acute glaucoma usually referred to as mydriatic glaucoma. This relationship is the basis of certain of the provocative tests used in the diagnosis of glaucoma.

Congestion of the Ciliary Body. Any increase in the volume of the vascular bed of the ciliary body will obviously result in a slight decrease in the chamber angle. Such congestion may be associated with neurovascular factors, with excitement, weeping, or other emotional experience; with upper respiratory disease, such as influenza, rhinitis, and sinusitis; and with the postoperative congestion following surgical operations in the region of the head. Another cause of congestion of the ciliary body is the action of the miotic drugs themselves. It is important only in the narrow-angle type of case.

CLINICAL VALUE OF GONIOSCOPY AND ITS LIMITATIONS

Gonioscopy has many practical applications. Unfortunately, too many men look to it for aid in the diagnosis of so-called primary glaucoma and, finding none, discard the technique entirely. The practical aspects of gonioscopy may be conveniently subdivided into two large groups: (1) its use in studying pathologic changes in the region of the limbus and chamber angle, and (2) its application in the study of glaucoma.

In studying disturbances in the limbal region and chamber angle gonioscopy permits direct observation of congenitally anomalous tissues, neoplastic tumors, cysts, foreign bodies, traumatic changes and their sequelae, and inflammatory changes and their sequelae. Moreu Morata in his *Manual de Gonioscopia* adds observation of nutritional-deficiency phenomena and the changes associated with diseases of the ocular fundi.

In studying congenital anomalies, gonioscopy permits direct observation of the presence or absence of persistent fetal mesoderm in cases of hydrophthalmos and megalocornea, the presence or absence of an iris-ciliary body bridge and defects of the zonular membrane in cases of congenital coloboma, and the condition and extent of the iris root in cases of so-called aniridia.

The presence of early neoplasms involving the ciliary body and iris root can be detected in some instances only by gonioscopy. An extension into the angle region may be the earliest sign of a mass involving the uvea anterior to the equator. The same may be said for cysts, whether traumatic, developmental, or parasitic. In cases of traumatic cyst, the origin and extent of angle involvement by the cyst can be detected only by gonioscopy. In one implantation cyst I was able to observe a direct communication between the cyst and a traumatic scleral defect at the limbus, so well covered by connective tissue and conjunctiva that slit lamp microscopy could be of no aid. The presence of a cilium in the angle associated with a pearl cyst may also be detected gonioscopically.

Traumata of the angle region, whether or not they are associated with the presence of foreign bodies, often cause the formation of anterior synechias. Their extent and the nature of any foreign bodies in the angle may be studied easily through the gonioscopic lens. In many instances the presence of hemorrhage in the anterior chamber precludes the examination of the angle. However, after absorption of the blood the source of hemorrhage may become visible gonioscopically. The presence of a traumatic cyclodialysis or tears in the zonule may likewise be detected only with this technique. A recent case of persistent hypotony of less than 5 mm. of mercury (Schiotz)

after a fist blow to the eye was explained when a traumatic cyclodialysis was found by gonioscopy in the temporal half of the angle. The crystalline lens was dislocated and the anterior chamber very shallow. By turning the patient's head to one side and then the other, so that the dislocated lens was tilted, half of the angle became visible.

Inflammatory lesions of subacute or chronic nature are those in which gonioscopy can be used most successfully. Lesions which involve the ciliary body and iris periphery can only thus be detected. Moreu Morata considers that gonioscopy can be of value in distinguishing between tubercular, syphilitic, septic, and focal inflammatory processes in the angle. He also believes that sympathetic ophthalmia can be distinguished gonioscopically from sympathetic irritation by the presence of injection and slight exudation in the ciliary body before the usual slit lamp findings appear. In my own experience I have been able gonioscopically to distinguish only the presence or absence of Koeppe nodule-like deposits in the angle in certain cases of chronic uveitis, without being clinically certain of their cause, although it was assumed that they were probably tuberculous or due to sarcoidosis. I consider gonioscopic findings in inflammatory disease as contributory clinical findings and like slit lamp findings, not necessarily diagnostic in themselves.

Aside from the pathologic changes in the angle described above, gonioscopy is valuable in cases of corneal opacity where some clear cornea remains peripherally. By this means one can determine the condition of iris, crystalline lens, and angle. It may thus be of great value in cases where keratoplasty is contemplated. In cases where tattooing is contemplated it may serve to recognize adherent iris.

Although gonioscopy has many applications in the conditions thus far discussed, it should be considered only as an aid in forming a complete picture. It has rather definite indications.

In its relation to the study of glaucoma, gonioscopy has its most interesting and valuable applications. First of all, gonioscopy has led to a better understanding of certain forms of glaucoma, especially acute (narrow-angle) glaucoma, and glaucoma following cataract removal. By permitting an evaluation of the angle depth, gonioscopy

permits sharper differentiation between acute (narrow-angle glaucoma) and glaucoma simplex. It is particularly helpful in distinguishing between congestive attacks of true acute ("primary") glaucoma and congestive episodes of "secondary" glaucoma associated with inflammation. The fact that the angle in true acute glaucoma is nearly always bilaterally shallow is an important aid in differentiation. The latter distinction is particularly helpful in diagnosing acute glaucoma due to intumescence of the crystalline lens, although bilateral cases do occur. Gonioscopy is often the only means of differentiating between the glaucoma occurring after cataract removal (due to complete synechia formation) and that due to iridocyclitis.

The most widely accepted use for gonioscopy is in the observation of sites of previous antiglaucoma operations as well as the sites of contemplated operations. It serves to reveal, in most cases, the causes of failure, thereby serving as a control of surgical technique. In addition it serves as a means of prognosis. The latter is particularly true after the cyclodialysis operation.

Gonioscopy in glaucoma, then, is very valuable in differential diagnosis and in consideration of prognosis and causes of failure as well as in observing the sites of antiglaucomatous operations. It is of no value in the diagnosis of glaucoma simplex where the problem of early diagnosis is so important. Nor does it explain the mechanism in most of the forms of glaucoma such as certain cases following trauma or uveitis. Nevertheless, gonioscopy has a definite permanent place in clinical ophthalmology.

Gonioscopic examination should not be attempted when the eyes are painful and inflamed. It usually cannot be used in acute glaucoma when the eyes are congested and the corneal epithelium is edematous. Clearing with glycerin is often surprisingly effective. In such eyes, however, the angles are often too shallow for examination even after the tension has been reduced to normal by miotics or surgical procedures.

Eyes in which mydriatic drugs have been effectively used are often difficult to examine gonioscopically, since the folded iris tissue

obstructs the view of the entire angle. It is well to instill miotics before examination in such cases.

NORMAL ANATOMY OF THE ANGLE OF THE ANTERIOR CHAMBER

The angle of the anterior chamber is extremely important from the viewpoints of pathology, physiology, and ocular surgery. It consists of the peripheral portion of the anterior chamber bounded posteriorly by the iris, and anteriorly by the corneoscleral trabeculum* and the most anterior portion of the ciliary body. The latter also forms the apex of the angle recess.

In order properly to understand the anatomy of the chamber angle, the widely held, fallacious idea that the root of the iris is inserted on the inner scleral surface, must be corrected. The iris is inserted on the anteromedial portion of the ciliary body at the point where the latter nears its insertion in the scleral spur and the posterior fibers of the corneoscleral trabeculum. Another important anatomical consideration is the extent of the opaque scleral wall covering the periphery of the anterior chamber and angle. Since the corneal margin is bevelled so that its shape is elliptical from in front and circular from behind, the amount of scleral wall between the corneal margin and the apex of the chamber angle varies considerably. La Grange gave the following figures: 1.75 mm. at the top, 1.45 mm. at the bottom, 1.0 mm. at either extremity of the horizontal meridian.

In embryonic life, when the anterior chamber is relatively small, the angle is entirely filled by a mesodermal reticulum which later undergoes progressive atrophy. Ultimately, at seven months, the process of atrophy results in a relatively deep recess bounded by a loose tissue meshwork. This "meshwork of the chamber angle" is divided into two portions which differ somewhat in histologic structure: the scleral meshwork or corneoscleral trabeculum and the uveal meshwork or pectinate ligament. The scleral portion arises in the deeper layers of the corneal lamellae just before the apparent termination of Descemet's membrane and runs back to the region

*The term trabeculum is used to designate the tissue as an organ in distinction from its component trabeculae.

of the scleral spur, becoming wider as it goes posteriorly. Especially in its posterior two thirds it contains a labyrinth of spaces which have direct access to the aqueous. The uveal portion consists of

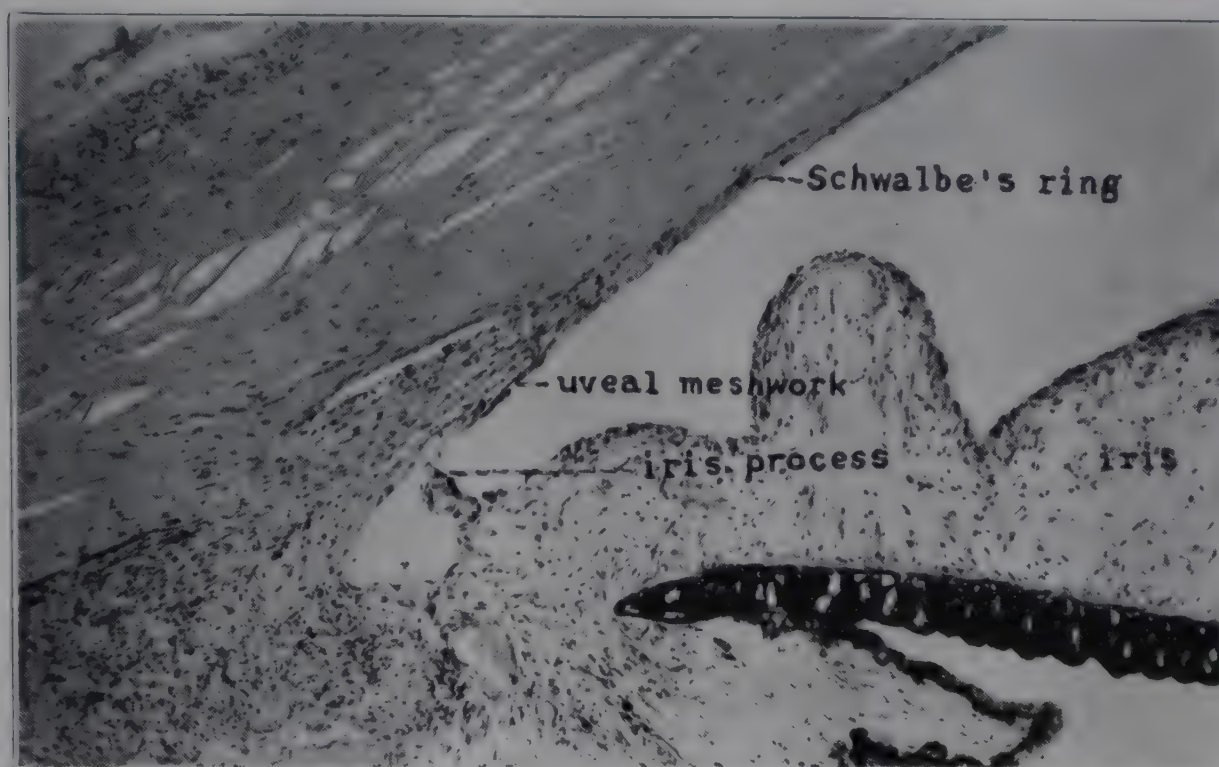


FIG. 277. Section of an atropinized eye, showing the bridging type of iris process and a portion of the uveal meshwork on the trabeculum.

rather delicate tissue which continues from the anterior border layer of the iris, lines the anterior surface of the ciliary body, and extends to cover the inner surface of the scleral meshwork. Some of this tissue is only one or two cell-layers thick, in particular, that portion covering the corneoscleral trabeculum. Other portions consist of comparatively thick, meridionally disposed bands which either line the recess of the angle or extend from the iris to the trabeculum, bridging the recess. These thicker portions are known, gonioscopically, as iris processes. The recess space partitioned off by a bridging iris process is the only human analogue of the spaces of Fontana of lower animals (Fig. 277).

In making a gonioscopic examination one begins at the iris on the side of the pupil away from the examiner and moves forward toward the insertion of the iris on the ciliary body and then across the angle recess to the trabeculum and the cornea. There is first seen an abrupt transition from the last roll of the iris to the anterior wall of the angle, usually obscuring most of the bow-shaped angle

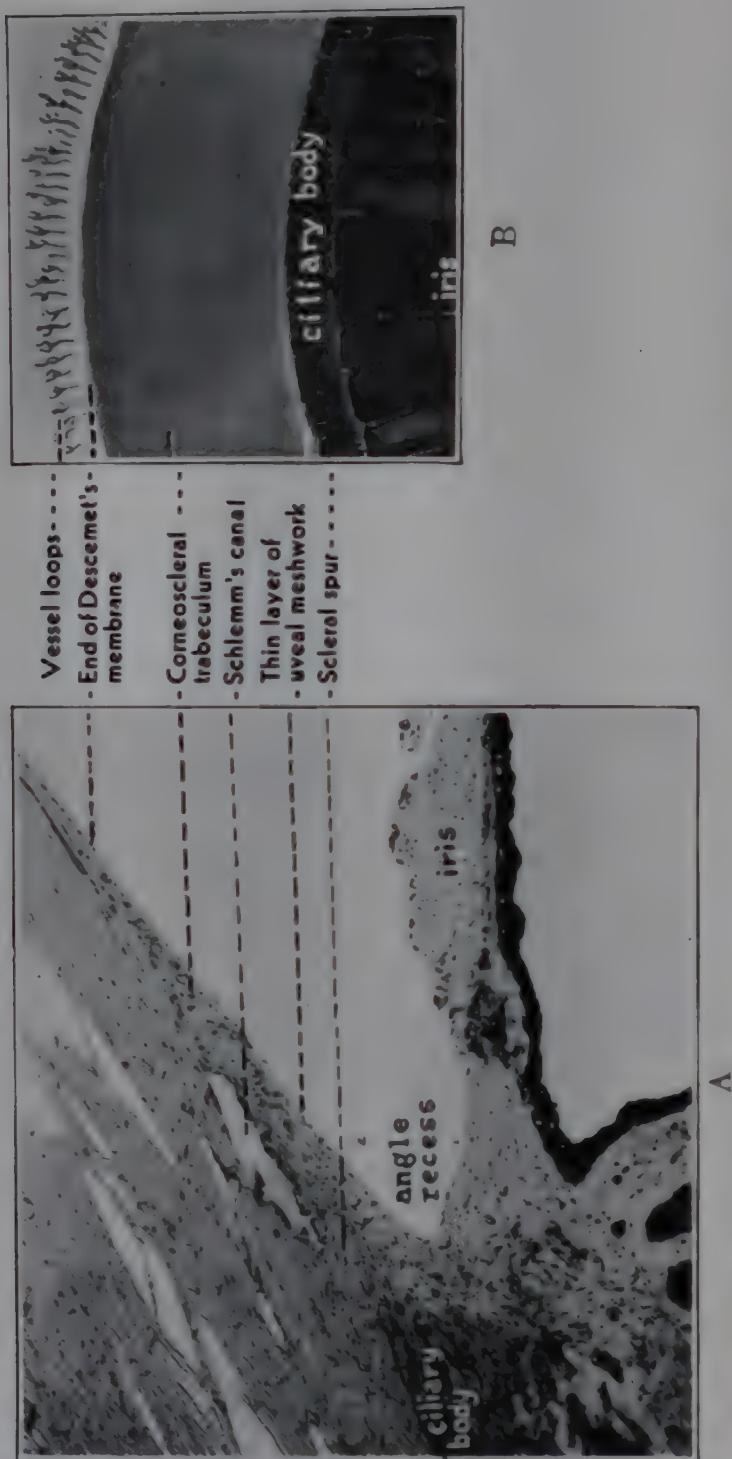


FIG. 278. Histologic (A) and gonioscopic (B) appearance of the normal chamber angle in a blue-eyed patient. The section is from an eye which was treated with physostigmine salicylate.

recess. Immediately above the transition line is the inner surface of the ciliary body as the latter passes toward its insertion in the scleral spur and the corneoscleral trabeculum. The gonioscopic ap-



FIG. 279. Lightly pigmented eye. The angle shows little differentiation between the ciliary body and the trabeculum. The arrows point out the width of the angle wall. A small amount of pigment is visible on the trabeculum. This and all other black and white photographs of the chamber angle are copies of kodachrome transparencies. The large white spot is the light reflex. The contact lens itself caused the dark zone in the uppermost portion of all the photographs of the angle.

pearance of the ciliary body and its inner covering of uveal meshwork depends on the intensity of pigmentation of the uveal tissues of the eye. In blue-eyed patients, ordinarily neither the ciliary body nor the iris processes stand out as they do in brown-eyed patients; therefore this area is a more or less diffuse light orange background on which gray or white iris processes may be seen (Fig. 278). In brown-eyed patients the iris processes, when prominent, stand out like a dark brown forest against the lighter brownish background of ciliary body. The processes are usually visible in negroid irides. Sometimes they cannot be discerned in light brown and blue eyes. In the latter, one frequently sees delicate blood vessels among the processes, apparently originating in the ciliary body.

There are only two important landmarks in the wall of the anterior chamber angle. The first is the anterior edge of the ciliary body. This corresponds to the position of the scleral spur which lies just behind the insertion of the ciliary body. The second landmark is the anterior edge of the corneoscleral trabeculum, which corresponds histologically to the place where Descemet's membrane ends (Fig.

279). This second landmark has been designated by gonioscopists as the "line of Schwalbe" because of the location here, also, of the anterior border ring of Schwalbe. However, since the latter is not

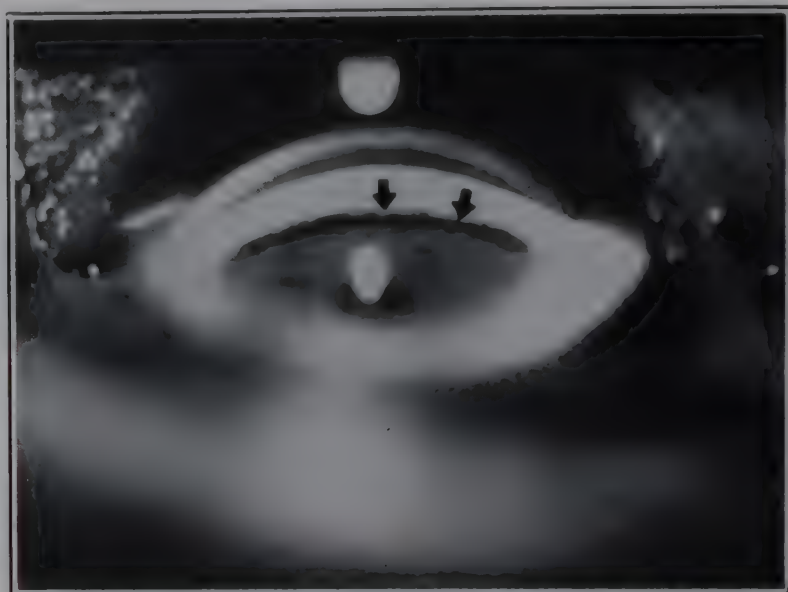


FIG. 280. The chamber angle in a normal, heavily pigmented eye showing iris process extending onto the trabecular wall. Arrows point to the end of Descemet's membrane.

always present and since it is not of any significance gonioscopically, I prefer to discard the term and designate the landmark as either "the termination of Descemet's membrane" or "the anterior edge of

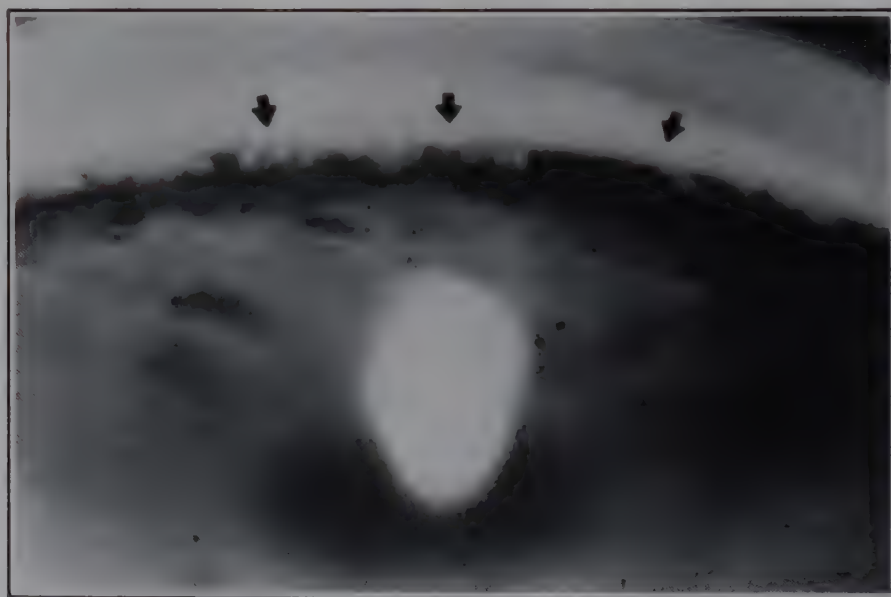


FIG. 281. Enlargement of angle shown in Fig. 280.

the trabecular band" (Figs. 280, 281). It must be emphasized that the important functional portion of the angle wall is the trabecular band sandwiched between the two visible anatomic landmarks mentioned above.

The extent of nontransparent tissue between the corneal margin and the angle apex from behind is only about one half to two thirds of the extent of the opaque zone from in front. From serial sections taken through the pupillary areas of three human globes * which were removed for tumors in the posterior portion of each, I measured the distances from the end of Descemet's membrane to the anterior margin of Schlemm's canal, from the anterior to the posterior ends of the canal, and from the posterior ends of the canal to the apex of the angle recess. The values (shown in Table VII) indicate that the width of the entire anterior angle wall is approximately 0.76 mm.

TABLE VII
MEASUREMENTS OF THE WIDTH OF THE CHAMBER-
ANGLE WALL

	EYE 1	EYE 2	EYE 3	AVERAGE
End of Descemet's membrane to Schlemm's canal	.343 mm. (.222-.444)	.377 mm. (.326-.459)	.393 mm. (.207-.503)	.371 mm.
Width of canal	.318 mm. (.252-.400)	.272 mm. (.207-.385)	.256 mm. (.207-.326)	.282 mm.
End of canal to recess	.062 mm. (0-.178)	.125 mm. (.030-.237)	.114 mm. (.030-.222)	.100 mm.
Total width of angle wall	.740 mm. (.610-.910)	.791 mm. (.607-.932)	.756 mm. (.577-.918)	.762 mm.

Between the two landmarks of the wall of the anterior angle lies the light tan colored corneoscleral trabeculum, a band about 0.6 to 0.7 mm. in width. Schlemm's canal lies deep to the posterior portion of the trabeculum.

Schlemm's canal is not normally visible during gonioscopic examination. However, it may be marked by a pink line when its contents include some blood. The factors which permit entrance of blood are apparently a decrease in intra-ocular pressure (Kronfeld),

* These sections were kindly loaned by Dr. Georgiana Dvorak-Theobald.

or inflammation. When a gonioscopic lens is applied to the eye, momentary pressure is exerted on it to cause a slight negative pressure between it and the eye. Coincidentally pressure is exerted on the conjunctival vessels under the rim of the lens. This pressure obstructs the aqueous veins, and may produce the blood-backflow phenomenon of Ascher. However, blood may be seen in Schlemm's canal under certain circumstances without the application of a gonioscopic lens by using Mizuo's method of gonioscopy in which the lids are held open by a speculum and the conjunctival sac filled with saline solution until the cornea is covered. By this means I have observed the presence of blood in the entire extent of Schlemm's canal in very soft eyes following penetration of the posterior segment by foreign bodies, where the cornea and anterior chamber were still entirely clear. Application of the gonioscopic lens in these cases caused no change in the angle picture.

In some eyes, usually with brown irides, extensions of the iris processes onto the trabecular tissue are seen. They may even extend onto the edge of the cornea. Sometimes only isolated islands of iris process tissue (uveal meshwork) may be seen on the trabeculum, not connected with the portion covering the angle recess and ciliary body.

Just forward of the end of Descemet's membrane one sees a band of meridionally situated vascular loops, branches of the rami recurrentes anteriores of the anterior ciliary vessels. Since the sclera overlaps the transparent cornea, these vessels cannot be seen from the outside but are visible gonioscopically. They are the loops which proliferate to form the deep corneal vessels which occur with inflammation such as parenchymatous keratitis.

Most anteriorly the inner concave dome of the cornea is seen in yellowish optic section.

In myopic eyes more of the ciliary body band is visible than in hyperopic eyes since the iris appears to be inserted relatively further back on the ciliary body. The anterior chamber consequently appears deeper in myopic than in hyperopic eyes. In many hyperopic

eyes the ciliary body and its anterior edge are obscured by the forward position of the iris. This often leads inexperienced observers to conclude that peripheral anterior synechias are present. In these

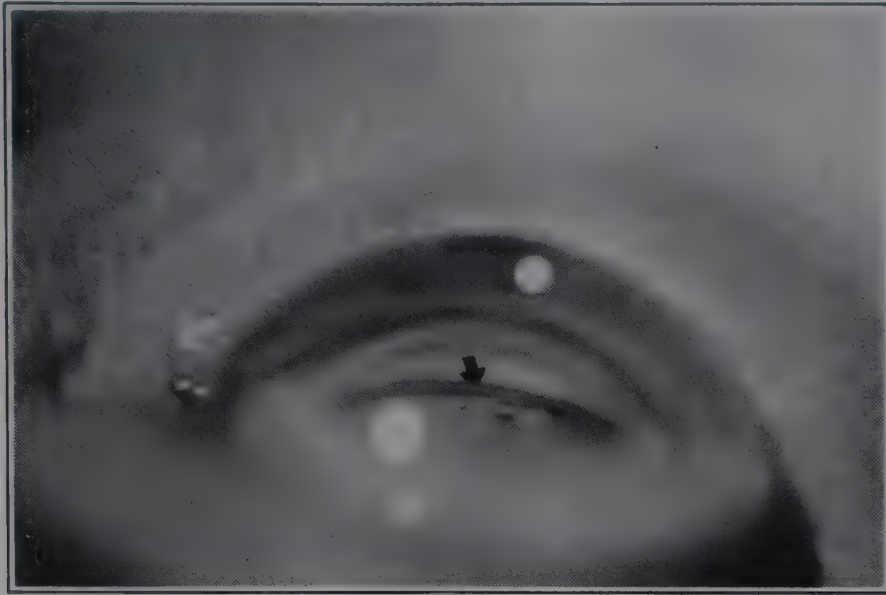


FIG. 282. Pigment deposited in angle following needling. The arrow points to the end of Descemet's membrane outlined by pigment.

cases it is usually necessary to partially transilluminate the angle and to examine it carefully with the binocular microscope to be certain that no peripheral anterior synechias are present.

In some middle-aged and senile individuals deposits of pigment granules in the spaces and the inner surface of the corneoscleral trabeculum covering Schlemm's canal give rise to the appearance of a brown trabecular ring. Finer pigment deposits are then usually present also on the end of Descemet's membrane and in the angle recess, particularly in the lower portion of the angle, probably due simply to gravitational factors. These pigment deposits are derived from the pigment epithelial cells of the iris and ciliary body (Fig. 282). Their significance is a matter of controversy. I believe that ordinarily such deposits are of little significance. One must be careful in describing trabecular pigment to distinguish between pigmented chromatophore cells and free pigment derived from degenerated pigment epithelial cells which is deposited on and in the trabecular wall from the aqueous.

Under the conditions of gonioscopic examination the view of the angle is considerably distorted by the contact lens itself as well as by

its position on the eye. The termination of Descemet's membrane (the line of Schwalbe) appears to protrude toward the anterior chamber. This illusion results from the fact that this line is the apex

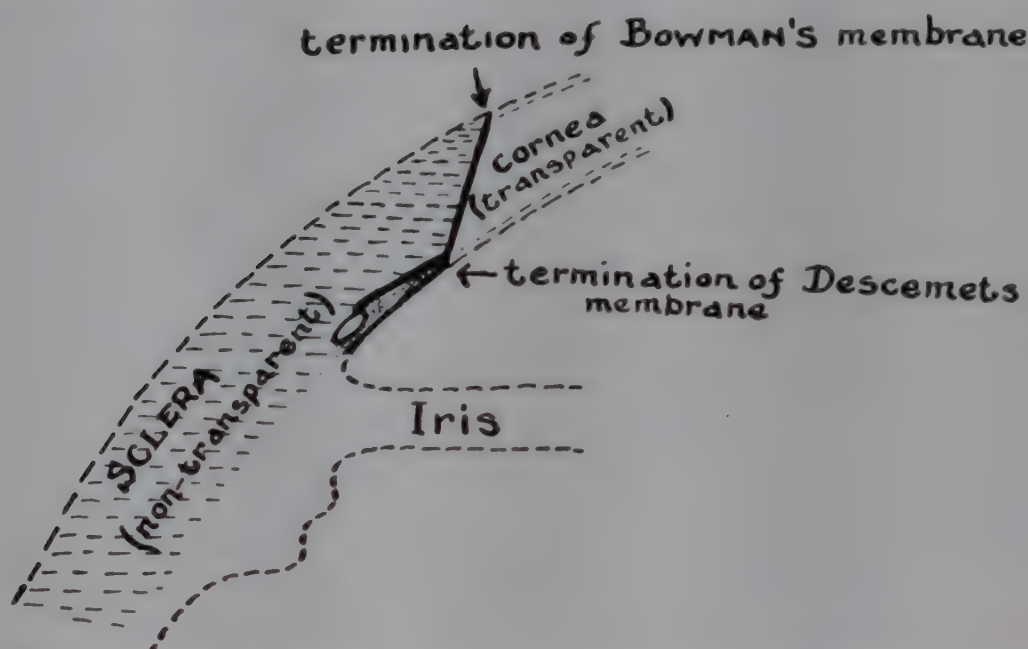


FIG. 283. The reason for the illusion of the protrusion of the end of Descemet's membrane into the anterior chamber. It is indicated by the apex of the nontransparent wedge outlined by the continuous heavy lines.

of an opaque angle formed by the inner trabecular surface and by the limbal boundary which extends from the end of Bowman's membrane to the apparent end of Descemet's membrane (Figs. 283, 284, 285).



FIG. 284. Plastic model of anterior segment of the eye with gonioscopic lens applied to show appearance of angle in various colors of iris.

In addition to this illusion, the trabecular wall appears to be considerably shorter than it actually is while the thickness of the limbus is exaggerated. When a trabecular pigment ring is present or when blood is seen in Schlemm's canal, it may appear in the anterior portion

of the trabeculum or in some cases in the vicinity of the termination of Descemet's membrane, instead of in its actual anatomic location, especially in eyes with narrow angles.

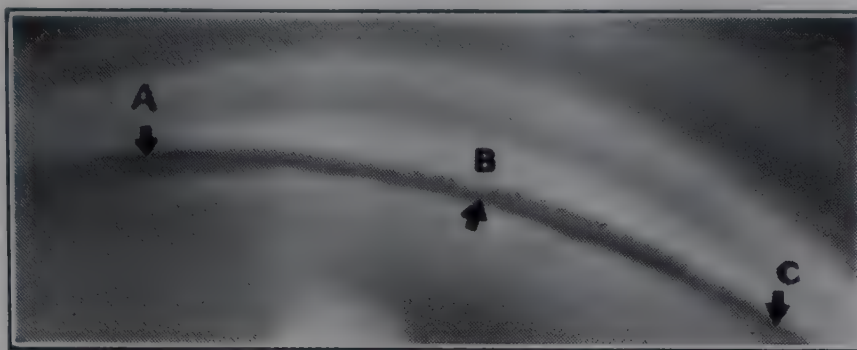


FIG. 285. Trabecular pigment ring in case of capsular exfoliation showing optical distortion from the gonioscopic lens. At *B* the ring is in its normal position. At *A* it appears on the level of the end of Descemet's membrane. At *C* the ring lies between the two other positions.

Miotics and mydriatics influence the visibility of the angle landmarks considerably. Pupillary dilatation with its associated increase in iris thickness causes a decrease in angle depth, while the use of miotics tends to stretch the iris tissues and to increase the depth.

CONGENITAL ALTERATIONS IN THE ANATOMY OF THE CHAMBER ANGLE

Earlier in this chapter it was pointed out that in embryonic life the space which later becomes the anterior chamber angle is filled by mesodermal tissue which undergoes partial atrophy and gives rise during the third to the fifth months to the anterior chamber and to the "meshwork of the chamber angle." Any interference with this process of atrophy may give rise to anatomic and physiologic disturbances in the angle, which may or may not manifest themselves in glaucoma. Among these anomalies are hydrophthalmia, congenital megalocornea (Fig. 286), congenital iris coloboma, and aniridia.

HYDROPTHALMIA

Association of anomalies of the angle and hydrophthalmia has been recognized since the latter was attributed to the persistence of mesodermal tissue in the angle by Horner, in 1880, and to the absence of Schlemm's canal by Cross, in 1896. Anderson in his com-

prehensive monograph on this condition attributed the glaucoma to one or more of the following causes:

1. Persistent or aberrant meshwork in the angle



FIG. 286. The Chamber angle in a case of megalocornea. Note that the mesodermal tissue continues to the end of Descemet's membrane. The trabecular wall is covered. S is the termination of Descemet's membrane, C the trabecular wall covered by mesodermal tissue, and I the iris root.

2. Poorly developed or absent canal of Schlemm
3. Posteriorly placed (fetal) canal of Schlemm
4. Rudimentary development of the scleral spur
5. Peripheral anterior synechias due to lack of differentiation or to post-inflammatory synechias.

In four cases of hydrophthalmia, the iris tissue in each case, instead of inserting on the ciliary body, continued along the angle to the region of the end of Descemet's membrane, obscuring any evidence of the landmarks of the angle wall. Obscuration of the landmarks did not appear to be due to synechias, since the iris markings continued over the entire tissue. In three of the cases in which the irides were blue, the reflection of the pigment epithelium from the ciliary body to the iris could be seen through this tissue. No conclusions can be drawn as to the presence or absence of Schlemm's canal from the gonioscopic examinations in these cases. In contrast to these cases of hydrophthalmia, cases of juvenile glaucoma, in which the ocular hypertension became manifest before the third decade, did not differ gonioscopically from the normal.

CONGENITAL MEGALOCORNEA

Two cases of congenital megalocornea were examined gonioscopically. Persistent mesodermal tissue continuous with the iris



FIG. 287. Angle at the root of a congenital coloboma of the iris.

was present, just as in the cases of hydrophthalmia. The possible relationship between hydrophthalmia and congenital megalocornea has been thoroughly discussed by Anderson.

CONGENITAL COLOBOMA OF THE IRIS

Two cases of bilateral typical coloboma of the iris with evidence of glaucoma were studied gonioscopically. In both cases the chamber angles were open and normal in each eye except for the areas of the colobomas where bridges of rudimentary iris tissue were present (Fig. 287). In one case the iris bridges were adherent to the trabecular tissue and the number of ciliary processes in the coloboma area were less than normal. The relationship between the glaucoma and the angle anomaly is not certain, since the angle in these cases had the same appearance that is found in nonglaucomatous iris coloboma cases. All these eyes with glaucoma had some decrease in the corneal diameters, which must also be considered in accounting for the glaucoma.

ANIRIDIA

Collins, in 1891, recognized the tendency of eyes with aniridia to develop glaucoma later in life. Anderson attributed the glaucoma

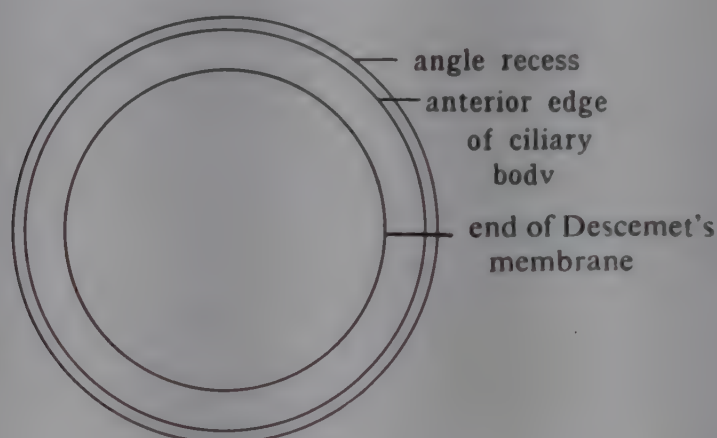


FIG. 288. Schematic representation of the relations of the angle recess, the anterior edge of the ciliary body and the end of Descemet's membrane.

to persistence of uveal meshwork or to adhesions between the iris stump and the cornea or the trabeculum. Of two patients with aniridia studied by me gonioscopically, one had glaucoma and the other did not. Both angles had the same appearance. An iris stump was present all around the angle in each case. In addition, persistence of a small amount of mesodermal tissue at the angle recess was noted in each.

PATHOLOGIC ALTERATIONS IN THE CHAMBER ANGLE GLAUCOMA

Gonioscopy finds its greatest value in the study of glaucoma. And since various etiologic entities are included in this group, they must be taken up separately. However, before discussing each, the role of peripheral anterior synechias should be considered. These are adhesions which form between the iris and the anterior angle wall, usually progressing from the apex of the angle toward the cornea. They may be either partial or total and they may be ciliary, trabecular or corneal, depending on their extent. When they are present, they are recognized by the obscuration of one or both landmarks of the anterior angle wall; namely, the anterior edge of the ciliary body and the end of Descemet's membrane. They may easily be dia-

grammed by indicating the extent of synechias on a background of three concentric circles representing the angle recess, the anterior edge of the ciliary body, and the end of Descemet's membrane,

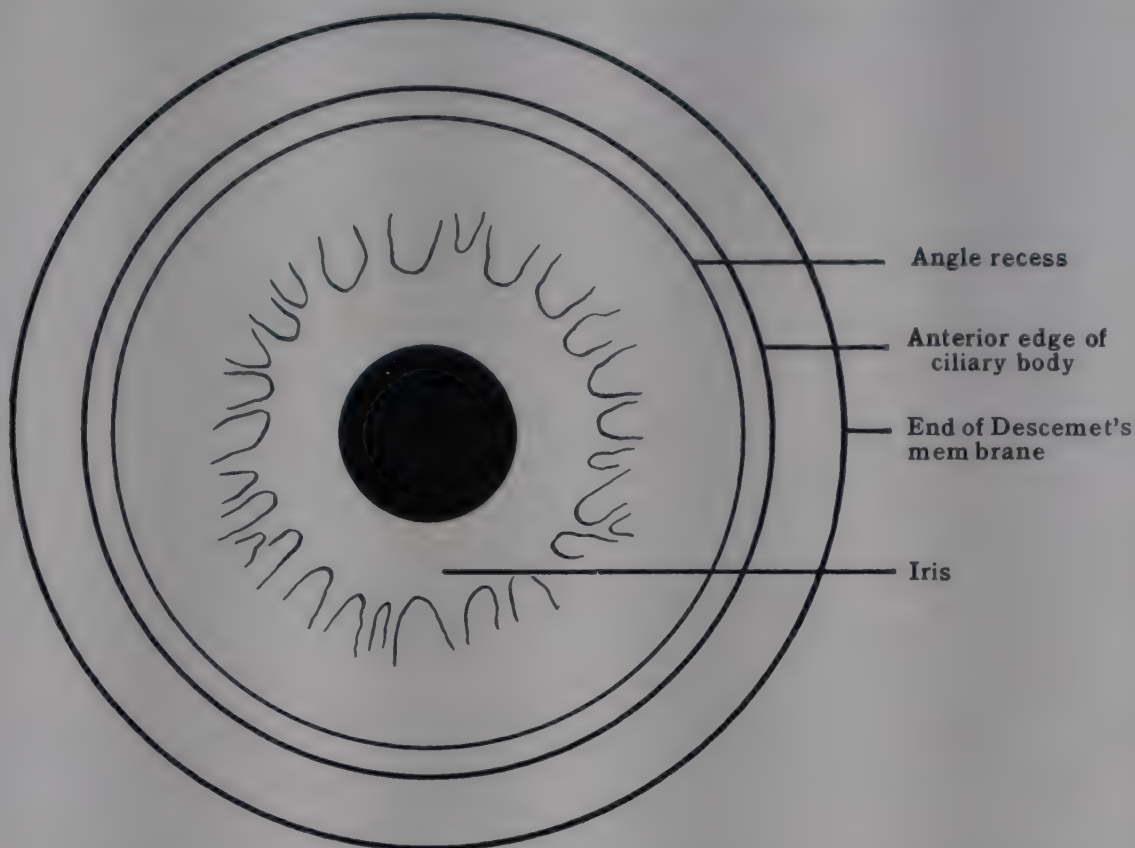


FIG. 289. Alternate schema for recording the location and extent of synechias.

respectively, from outside in. Figure 288 shows such a diagram, as suggested by Kronfeld.

Another method of recording the location and extent of synechias is by the use of a diagram in which the anterior wall of the angle is considered to be folded back into the same plane as the iris (Fig. 289). Troncoso and I have both used this method. Either will serve the purpose.

Knies and Weber independently discovered peripheral anterior synechias in eyes which were enucleated after long-standing glaucoma and assumed that the synechias were the cause of glaucoma. Troncoso refuted this idea and showed that the synechias were really the result rather than the cause of the glaucoma. The truth of this, as far as shallow angle and simple glaucoma is concerned, has been substantiated by all subsequent observers.

Gonioscopic experience establishes one outstanding clinical fact; that is, peripheral anterior synechias are the result of hyperemia and

edema of the ciliary body; they do not occur in any form of glaucoma unless congestion is present. That this is true is confirmed by the experimental work of Friedenwald and Pierce who produced acute glaucoma in dogs by injecting histamine into the vitreous.

Narrow-Angle Glaucoma

The congestive phases of acute glaucoma and that clinical subvariety of acute glaucoma which is known as "chronic congestive glaucoma" belong to the category of narrow-angle glaucoma. The narrowness of the chamber angles and the facts concerning the depth of the chamber angle discussed under "goniometry" suggest that this is a mechanical form of glaucoma due to closure of the angle by the iris root. At any rate, gonioscopic observations support the opinion that this type of glaucoma should be classified separately from chronic simple glaucoma.

Salzmann, the first to use gonioscopy in the study of glaucoma, found the chamber angles partly closed in three eyes with "congestive glaucoma." Later, Troncoso in studies of "acute or subacute congestive glaucoma," observed 7 cases (26 per cent) in which the angles were open, 12 (46 per cent) in which they were partly closed, and 7 (26 per cent) in which they were entirely closed. In studying "chronic congestive glaucoma" he observed 2 cases in which the angles were open, 2 in which they were partly closed and 4 in which they were totally closed. He concluded that in the beginning of acute glaucoma the angle is open and that it closes later if the attack is severe or persistent. Thorburn found synechias in 3 of 6 cases with "congestive glaucoma." Werner found the angle closed by adhesions in one eye with acute glaucoma and partly closed in two with "chronic congestive glaucoma." McLean, in 21 cases of "chronic congestive glaucoma," found no synechias in 4, minor ones in 6, and extensive ones in 11.

Of my own series of 68 eyes with acute glaucoma, the tension was reduced to normal within one to three days after the onset of the attack in 13 instances (Table VIII). In 9 of the 13 eyes the angle was deep enough to be visible gonioscopically; 6 of these had

TABLE VIII
ACUTE GLAUCOMA RELIEVED BY MIOTICS

PATIENT	ANGLE BEFORE ATTACK	DURATION OF ATTACK	ANGLE AFTER NORMALIZATION WITH MIOTICS
1. M. C.	Open	1 day	Open
2. B. D.	3 days	Open; trabecular adhesions in places
3. T. R.	2 days	Too shallow
4. L. S.	Open	1 day	Open
5. A. T.	1 day	Open
6. R. H.	2 days	Too shallow
7. J. K. (RE)	1 day	Open
8. J. K. (LE)	1 day	Open
9. A. B.	1 day	Too shallow
10. E. B.	2 days	Open $\frac{2}{3}$; too shallow $\frac{1}{3}$
11. A. P.	2 days	Small upper temporal area open; later tension rose
12. M. K.	1 day	Open
13. A. K.	3 days	Open 12 to 3 o'clock; closed elsewhere

entirely open angles, and 3 had partly open angles. The open angles were all in eyes in which the attack had been relieved within twenty-four hours of its onset. In the 3 with partly open angles the attack had lasted from forty-eight to seventy-two hours. One of these had an open area extending about 25 degrees along the circumference of the angle. In this eye the tension had been reduced to normal by miotics but subsequently rose again. The border of the pupil, at the time the tension was found to be normalized, was flattened in the area corresponding to the open portion of the angle. For the remaining 55 eyes in this series, miotics were used from two days to seven months without relief of the glaucoma.

All evidence indicates that the angle is normal although narrow before an attack of acute glaucoma, but is closed during the attack. If miotics are used early, before the adhesions have become firm, at least a portion of the angle is opened and the tension is reduced to

normal. If the attack persists for more than two or three days, the adhesions have usually become too firm for miotics to be of any avail. Operations must then be resorted to.

In a series of 33 eyes with "chronic congestive glaucoma" I found the angle too shallow for observation in 13, narrow but open in 19 and entirely closed in one. In this group the same process in a milder form than in the eyes with acute glaucoma probably goes on. Synechias form if the congestive phase lasts long enough or is repeated sufficiently often.

When firm synechias form in the congestive phases of narrow-angle glaucoma, only rarely can they be detached with the use of miotics. In these cases shreds of the anterior border layer of the iris remain attached to the trabecular wall.

It would not be amiss here to explain briefly the terminology which I am using in the classification of glaucoma. Terms such as congestive and noncongestive or their synonyms are either being avoided or marked by quotation marks because of the fact that these terms not only are nonessential to the classification but are actually the cause of much confusion. Elsewhere,²⁸⁸ I have described the findings in a study of 49 cases (74 eyes) with acute glaucoma which were investigated to determine the exact onset of congestion in relation to the onset of the glaucoma symptoms. In that series I found that the majority of cases, whose onset was precipitated by mydriasis, accommodative effort, lenticular intumescence, or ciliary body congestion, began without vascular congestion, but after a period varying from several hours to days, the eye became red because the vascular system of the eye could no longer tolerate the acute ocular hypertension. If seen before the onset of congestion, a patient with acute glaucoma cannot be diagnosed as having acute congestive glaucoma; the condition should be called acute glaucoma, noncongestive phase. If seen when vascular decompensation is present, the diagnosis should be acute glaucoma, congestive phase.

In recent years there has been a tendency to substitute the term *narrow-angle glaucoma* (Barkan) for the previous classification of acute and chronic congestive glaucoma. This is a definite advance in

our thought and helps avoid the inclusion of a description of the vascular reaction of the eye in the diagnosis as discussed above. Yet the terms are, strictly speaking, not synonymous, since narrow-angle glaucoma covers a wider range of cases:

1. Classical "acute congestive glaucoma"
2. "Chronic congestive glaucoma"
3. Dilatation glaucoma
4. "Preglaucoma"
5. Acute glaucoma due to lenticular intumescence
6. Acute glaucoma due to dislocation of the lens into the anterior chamber
7. Mixed glaucoma (including acute glaucoma as one form)

The first four conditions should be considered together under the term acute glaucoma. The others should be considered separately.

The following classification of the glaucomas is based on clinical and gonioscopic observations.

I. "*Primary Glaucomas*"

A. Glaucoma Simplex (Chronic Simple Glaucoma)

1. Noncongestive phase
2. Congestive phase (rare)

B. Glaucomas caused by anatomic and developmental anomalies

1. Congenital glaucomas
 - a. Hydrophthalmos
 - b. Glaucomas associated with aniridia, or neurofibromatosis
2. Juvenile glaucoma
3. Acute (narrow-angle) glaucoma—due to anatomic plus physiologic angle-narrowing factors which lead to mechanical obstruction of trabecular spaces by iris. This includes acute glaucoma associated with microcornea
 - a. Noncongestive phase—including "dilatation glaucoma"
 - b. Congestive phase—classical "acute congestive glaucoma" and the recurrent form called "chronic congestive glaucoma" in the older classification

II. "*Secondary Glaucomas*." Each may be subdivided into a noncongestive and a congestive phase. Some never enter the congestive phase.

A. Secondary glaucomas due to mechanical blockage of the trabecular spaces

1. Obstruction by iris
 - a. Acute secondary glaucoma due to lenticular intumescence
 - b. Acute secondary glaucoma due to dislocation of the lens into the anterior chamber
 - c. Glaucoma following operation for cataract—*aphakic obstructive glaucoma*—due to delayed reformation of the anterior chamber
 - d. Glaucoma associated with essential progressive atrophy of the iris
2. Obstruction of the trabecular spaces by particulate matter
 - a. Glaucoma capsulare
 - b. Pigmentary glaucoma
 - c. Glaucoma due to obstruction by lens particles
 - d. Glaucoma due to tumor growth
 - e. Glaucoma due to cellular debris associated with active or healed iridocyclitis
- B. Secondary glaucomas due to lack of communication between the anterior and posterior chambers
 1. Secondary glaucoma due to seclusion of the pupil
 2. Secondary glaucoma due to total posterior synechia
- C. Secondary glaucomas probably due to overproduction of aqueous as a result of irritation of the ciliary processes
 1. Glaucoma associated with posterior dislocation of the lens as that latter touches ciliary processes
 2. Cyclitis and anterior choroiditis
- D. Secondary glaucomas due to obstruction of venous drainage
 1. Experimental and clinical glaucoma due to vortex vein obstruction
 2. Secondary glaucoma in pulsating exophthalmos
- E. Secondary glaucomas due to newly proliferated anastomotic vessels involving the Schlemm's canal mechanism in rubeosis iridis (diabetic and arteriosclerotic) and following occlusion of the central retinal vein
- F. Secondary glaucoma resulting from trauma
- G. Secondary glaucoma associated with epidemic dropsy
- H. Secondary glaucoma associated with choroidal angiomatosis

Glaucoma Simplex (Chronic Simple Glaucoma)

The depth of the anterior chamber in cases of simple glaucoma does not differ from the normal; this was shown by Rosengren and corroborated by Gradle and Sugar. As a matter of fact, in early

and moderately advanced cases there is nothing in the appearance of the angles to distinguish them from normal. In certain far advanced cases of simple glaucoma and absolute simple glaucoma which have entered a mild congestive phase, peripheral anterior synechias do form. Sometimes the onset of vascular decompensation in these cases is ushered in with severe symptoms and signs simulating an attack of acute glaucoma. Erroneous diagnoses of acute glaucoma are frequently made in these cases, especially when the histories are not adequate. The correct diagnosis should be "glaucoma simplex (in the) congestive phase."

Salzmann examined one eye with simple glaucoma and found the angle entirely open. Troncoso, in observing 34 eyes with "non-congestive glaucoma," found 56 per cent with open angles, 38 per cent with partly closed angles, and 5.8 per cent with completely closed angles. Thorburn, in nine cases of simple glaucoma, found 6 angles entirely open and 3 partly closed. Werner found that 20 of 28 eyes had open angles and the remainder had synechias. Barkan found the angle open in 60 per cent of cases of this type of glaucoma. McLean, in 66 simple glaucoma cases, found no synechias in 74.3 per cent, minor synechias in 22.7 per cent, and extensive synechias in 3 per cent.

I examined a series of 344 eyes with chronic simple glaucoma and found the angle open in 337 (97 per cent). This series was divided into four groups, according to the extent of damage to the visual fields: (1) cases of early involvement, in which the visual fields showed minimal change; (2) cases of moderately advanced disease, in which the fields varied in constriction up to 10 degrees from the fixation point; (3) far advanced cases, in which the fields were within the 10 degree circle; and (4) cases of absolute glaucoma. In the first two groups (314 eyes) there were no peripheral anterior synechias. In the third group (10 eyes) 3 had open angles, 4 had partly open angles, and 3 had completely closed angles. Of the 20 eyes with absolute glaucoma, the angles were open in 7, partly open in 6 and entirely closed in 7.

It must be concluded that peripheral anterior synechias occur

in simple glaucoma only in far advanced cases or cases of absolute glaucoma simplex. A congestive phase is apparently necessary to bring about the formation of synechias.

Barkan attributed considerable significance to trabecular sclerosis and pigment granule blockage of the trabecular spaces as etiologic factors in simple glaucoma. No subsequent observers have recognized trabecular sclerosis gonioscopically. I found pigment granules, however, in 50 per cent of 199 eyes with simple glaucoma. Since pigment is also present, though less frequently, in the normal non-glaucomatous eyes of the middle-aged and senile individuals, it must be considered of relatively little importance except in those few cases in which dense trabecular pigment deposits are present. It is conceivable that in these cases the trabecular spaces may be so filled with pigment that mechanical access of the aqueous to the osmotic influence of the fluid in Schlemm's canal is hindered.

Glaucoma Capsulare

In addition to the slit lamp findings in the anterior segment and vitreous in eyes with this most interesting type of glaucoma, gonioscopic technique adds considerably to our knowledge and understanding of the disease. Because of the fact that the slit lamp is not routinely used in the examination of glaucomatous eyes, cases of glaucoma capsulare are usually erroneously diagnosed as glaucoma simplex. Also, because the tension is usually not high, many cases of glaucoma capsulare remain unrecognized.

Glaucoma capsulare is a complication of exfoliation of the zonular lamella of the lens capsule and differs from the latter only in the presence of ocular hypertension and its manifestations. Since both conditions have the same appearance in slit lamp and gonioscopic observations, the description of the angle will include both.

Exfoliated particles of the zonular lamella on the trabeculum were recognized histologically by Bey and gonioscopically by Barkan and by Gradle and Sugar. Large particles sometimes stand out as they lie on the end of Descemet's membrane or on the trabeculum. Finer particles mixed with pigment dust give the ciliary body and the

trabeculum a faintly veiled appearance in lightly pigmented eyes. The pigment deposit in the trabecular spaces over Schlemm's canal produces a characteristic trabecular pigment ring which develops



FIG. 290. Deposit of pigment on the trabecula in a case of capsular exfoliation. The area below the pigment line is the ciliary body. The arrows point to the pigment line.

in all cases of capsular exfoliation or of glaucoma capsulare (Fig. 290). The pigment granules are derived from the pigment epithelium of the iris which is traumatized by its constant movements against the roughened zonular lamella. In 11 cases of unilateral capsular exfoliation, the uninvolved eye in 9 showed no pigment ring, while in every instance the eye with exfoliation showed a typical ring.

In eyes in which iridectomy has been performed, involvement of the equatorial zonular lamella and the zonule fibers, and granules of exfoliated material on the ciliary processes can be seen gonioscopically. The equatorial zone involvement consists of whitish granules in the area where the zonule fibers insert into the lens capsule. This was described in 1940 by Gradle and Sugar and shortly afterward by Irvine. Opacification of the individual zonule fibers was noted at the time of the first description of equatorial exfoliation. Trantas, in 1926, noted exfoliations on the zonule fibers. These may be deposits of exfoliated material from the lens capsule or from the fibers themselves. Irvine believes the latter to be true.

An interesting result of the presence of capsular exfoliations on the surfaces of the wall of the angle in cases of glaucoma capsulare before and even after surgical operations in these eyes, is the relative

absence of peripheral anterior synechias. Rare cases do occur, of course, in which synechias appear as a result of vascular decompensation in an eye with absolute glaucoma capsulare. The absence of any considerable tendency to synechia formation is probably attributable to the insulating effect of the capsular exfoliations.

Many surgeons consider that removal of the crystalline lens from eyes with glaucoma capsulare will relieve the condition. It is true that removal of the lens will prevent further exfoliation. However, the blockage of the angle already present is not affected, so that further treatment of the glaucoma is usually necessary. Of 16 eyes with glaucoma capsulare from which the lenses were removed, 14 continued to have ocular hypertension of the same degree or only a slightly less degree, than before operation. Only 2 of the 16 required antiglaucoma surgery. Twelve others were controlled with miotics.

The depth of the angle in cases of glaucoma capsulare is nearly always the same as the normal depth. Occasionally one encounters a patient with shallow chambers and capsular exfoliation or glaucoma capsulare just as one may occasionally find a case of glaucoma simplex in an eye with a shallow chamber and angle which later may have true acute glaucoma superimposed. In these cases the presence of the two conditions in the same eye is purely coincidental.

Glaucoma Associated with Lenticular Intumescence

There is no anatomic difference between the angle in this type of glaucoma and that in the ordinary type of acute glaucoma. Lenticular swelling produces narrowing or actual blockage of the angle by the iris root. If the angle is only narrowed, any other factor which tends to decrease the angle depth will precipitate acute glaucoma. These factors have been discussed in the section on goniometry. I provoked an acute increase in ocular tension in a patient with an intumescent lens with one drop of 1 per cent paredrine hydrobromide solution. Refraction previously had shown the affected eye to be myopic 14 diopters. The chamber had been normal in depth before the lenticular swelling took place. The tension before instillation of the paredrine solution was 17.5 mm. of mercury (Schiotz).

Forty minutes later the pupil had dilated from 4 mm. in diameter to 8 mm., and the tension had risen to 48 mm. of mercury. The angle was then too shallow for gonioscopy. A drop of 2 per cent pilocarpine nitrate solution rapidly reduced the tension to normal.

In three patients with acute glaucoma and lenticular intumescence closure of the chamber angle was present. Miotics were effective in relieving the hypertension in each case prior to operation for cataract. In one case the preoperative use of cocaine hydrochloride solution precipitated a second attack.

Glaucoma Associated with Dislocated or Subluxated Lenses

Sixteen eyes with this condition were examined gonioscopically. Thirteen had wide open angles. In two with subluxated lenses which apparently impinged on the back of the iris root below, a localized narrow synechia was present. One other eye had fine trabecular adhesions all around the angle. The lack of extensive synechias in these cases indicates that the glaucoma is not due to angle blockage. The theory that it is due to irritative hyperactivity of the ciliary processes with consequent increased production of aqueous seems reasonable.

Hemorrhagic Glaucoma

Hemorrhagic glaucoma following occlusion of the central retinal vein. In patients with obstruction of a venous branch or with partial obstruction of the central vein, glaucoma does not develop in the involved eye and the angles appear entirely normal. When glaucoma develops in cases of complete occlusion of the central vein, the angle remains open at first but is considerably congested. A short time later the angle is found to be completely closed. Many new anastomotic vessels are then seen at the point where the iris becomes adherent to the cornea.

Diabetic rubeosis iridis. Kurz described the appearance of the chamber angle in this condition in 1939. In the ten eyes observed by me the angles had the same appearance as those in cases of hemorrhagic glaucoma following obstruction of the central vein. As in the

cases described by Kurz, the angles were completely closed and covered by fine anastomotic vessels in the old cases but open and showed anastomotic vessels entering Schlemm's canal in 2 early cases.



FIG. 291. Traumatic cyclodialysis in a patient with hypotony following injury.

Both types of hemorrhagic glaucoma are associated with hemorrhages in the retina and vitreous. The nutritional disturbance in each of these conditions, associated with hypoxia and an accumulation of toxic metabolic products, probably stimulates the formation of the new vessels which destroy the Schlemm's canal mechanism.

TRAUMATIC ALTERATIONS IN THE CHAMBER ANGLE

In cases of perforating trauma involving the angle and its neighboring structures, the alterations in the angle depend on the duration of collapse of the anterior chamber and the degree of inflammatory and exudative reaction. Usually extensive synechias appear in the particular areas of reaction. Only if most of the angle is thus involved does glaucoma result.

With nonperforating trauma, the acute reaction may subside completely with little or no subsequent damage to the eye, or it may be followed by glaucoma. Five cases with the latter complication were examined gonioscopically. In none of these was there any evidence of persistence of the acute traumatic inflammatory reaction. The angle was entirely open in each case. No conclusions as to the cause of the increased tension could be drawn.

Occasionally nonpenetrating trauma is followed by hypotony. In two instances which I have seen, a broad cyclodialysis was present (Fig. 291).



FIG. 292. Tumor nodule in angle in patient with proven sarcoidosis.

TUMORS, CYSTS, AND FOREIGN BODIES IN THE ANGLE

Gonioscopic technique is the only means of observing the extent of involvement of the angle by any suspected pathologic condition. Diagnosis of neoplasms may be made earlier in some cases if this

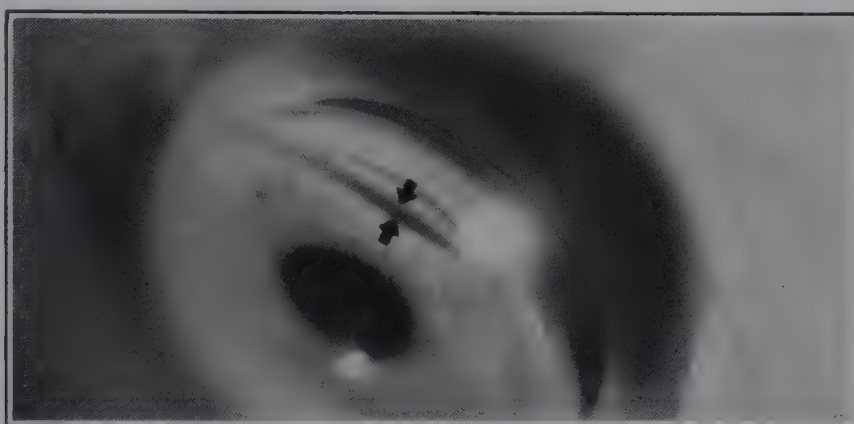


FIG. 293. Iridodialysis due to malignant melanoma of ciliary body involving angle.

method of examination is resorted to (Figs. 292, 293). Their origin, extension, or metastasis may thus be determined. The nature of small cysts of the iris and their increase in size are best observed in this way.

Bruce and others have reported the use of gonioscopy to localize foreign bodies in the anterior chamber. Only in this manner can their presence in the angle be ascertained, especially if they are multiple small particles of glass or other transparent material.

INFLAMMATORY ALTERATIONS IN THE CHAMBER ANGLE

The degree and extent of inflammatory reaction determine the amount of involvement of the chamber angle in keratitis, limbal

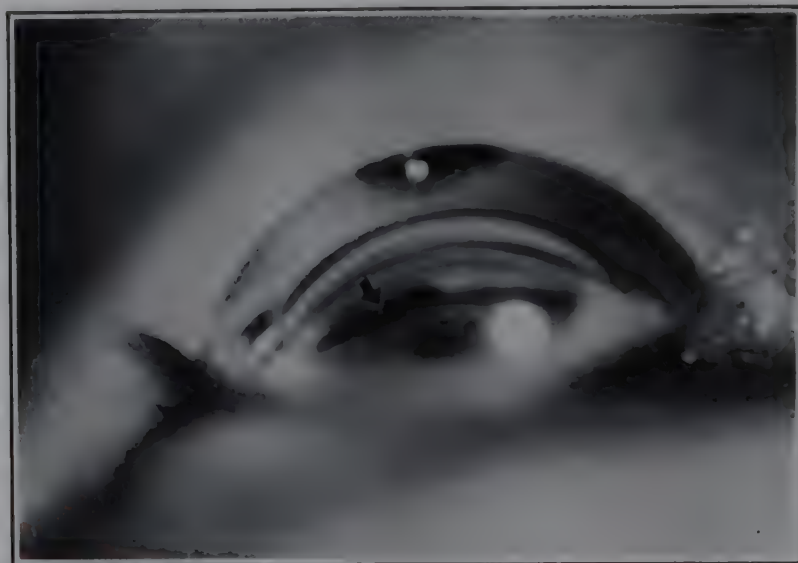


FIG. 294. Broad anterior synechia in secondary glaucoma following iritis with exudate in angle.

scleritis, iridocyclitis, and cyclitis. Cases of iris bombé are obviously associated with obliteration of the angle and cannot usually be studied gonioscopically. In early, active, acute or chronic iridocyclitis the angles are usually normal. When fibrin and exudate are present they can usually be seen with the slit lamp and microscope and gonioscopy need not be resorted to. Weeks or months after the subsidence of such inflammation, the angle is usually partly or entirely open, but it is covered with spots of pigment and cellular debris. When synechias are found, they are circumscribed and appear to result from the organization of fibrinopurulent exudate (Fig. 294). It is conceivable that organization of exudate within the trabecular spaces mechanically hinders the access of aqueous to the walls of Schlemm's canal.

POSTOPERATIVE ALTERATIONS IN THE CHAMBER ANGLE

Unquestionably the most gratifying use of gonioscopic technique is in the study of the results of glaucoma operations and of cases of glaucoma following cataract operations. A series of over 300 anti-glaucoma operations and 52 glaucomatous eyes following cataract extraction were studied gonioscopically. A third of the group which

had glaucoma of the narrow-angle and simplex types had also been examined preoperatively.

Glaucoma Following Surgical Treatment of Cataract

Numerous clinical studies of glaucoma following cataract removal have been published since Bowman's first report in 1865. From histologic and clinical evidence Czermak, in 1898, concluded that this type of glaucoma was due to prolonged absence of the anterior chamber as a result of delayed closure of the wound. Histologically, he found the iris adherent to the periphery of the cornea.

In 1940, I reported the gonioscopic findings in a series of 30 cases of glaucoma following cataract removal. The angle was completely closed in 29. In the remaining one, in which a small portion of the angle was open, the tension was reduced to normal with miotics. The histories in many of the cases indicated a prolonged postoperative period during which the anterior chamber was collapsed, associated in some instances with recognized choroidal detachment or prolapse of the iris. I concluded that complete closure of the chamber angle was probably the cause of this type of glaucoma. This conclusion was later corroborated by Kronfeld and Grossmann in a study of the relation between delayed restoration of the chamber, the formation of peripheral anterior synechias, and the occurrence of glaucoma.

Further study of cases of glaucoma following cataract operation has permitted subdivisions of these cases into two groups: those in which the glaucoma was first noted after discission of an after-cataract, and those in which the glaucoma followed removal of the crystalline lens. A series of 52 eyes was studied. In each case the tonometric tension was normal prior to operation. Cases in which uveitis was present and those in which the interval between the cataract operation and the development of ocular hypertension was more than a year, were excluded, since some of these may have been cases of glaucoma simplex.

Of the group in which the glaucoma followed the removal of the lens (40 eyes), all but two had peripheral anterior synechias

TABLE IX

GLAUCOMA FOLLOWING OPERATIONS FOR CATARACT

PATIENT	TYPE OF OPERATION	INTERVAL BEFORE FORMATION OF ANTERIOR CHAMBER	INTERVAL BETWEEN CATARACT OPERATION AND FIRST EVIDENCE OF GLAUCOMA	CHAMBER ANGLE	MEANS USED TO RELIEVE GLAUCOMA
1. P. N.	Intracapsular	5 days; collapsed on 10th day; choroid detached	1 month	Obliterated; ointment globule in anterior chamber	Part of oil removed through keratome incision; operation
2. J. M.	Extracapsular	4 flaps over iris prolapse	2 months	Grossly obliterated	Operation
3. S. L.	Intracapsular	3 flaps over iris prolapse	2 months	Grossly obliterated	Uncontrolled
4. E. B.	Extracapsular	Hyphemia 6 weeks	6 weeks	Obliterated	Uncontrolled
5. J. C.	Extracapsular	1 day; cortical remnants	7 months	Obliterated	Operation
6. J. B.	Extracapsular	Prolapse	2 months	Obliterated	Uncontrolled
7. T. L.	Intracapsular	7 days; prolapse; anterior chamber collapsed on 10th day on removal of suture	9 weeks	Trabecular adhesions below; closed elsewhere	Operation
8. M. H.	Extracapsular	5 days; prolapse	Occasional tension 28; once 37	Obliterated	Filtering cicatrix?
9. A. K.	Extracapsular	3 days	5 weeks	At least $\frac{2}{3}$ trabeculum covered by iris all around	Operation
10. H. G.	Extracapsular	13 days	5 weeks	Obliterated	Operation
11. M. M.	Extracapsular	Trauma; hyphemia 7th day	6 weeks	Obliterated	Operation
12. M. M. (R) .	Extracapsular	6 days	3½ months	Obliterated	Miotics
13. M. M. (L) .	Extracapsular	12 days	6 weeks	Obliterated	Operation
14. L. R. (R) ..	Intracapsular	4 days	1 month	Obliterated	Filtering cicatrix?
15. L. R. (L) ..	Intracapsular	4 days; collapsed on 6th day; reformed 11th day; choroid detached	6 weeks	Small opening 7 to 8 o'clock; obliterated elsewhere	Filtering cicatrix?
16. D. S.	Intracapsular	14 days; choroid detached	3 months	Obliterated except at 5 : 30 o'clock	Miotics
17. M. M.	Extracapsular	5 days; choroid detached 8 days	10 weeks	Obliterated	Operation
18. A. S.	Extracapsular	19 days	7 months	Obliterated	Operation
19. H. S.	Intracapsular	8 days	3 months	At least $\frac{2}{3}$ trabecula below obliterated; obliterated above operation	Operation

TABLE IX — *Continued*

GLAUCOMA FOLLOWING OPERATIONS FOR CATARACT

PATIENT	TYPE OF OPERATION	INTERVAL BEFORE FORMATION OF ANTERIOR CHAMBER	INTERVAL BETWEEN CATARACT OPERATION AND FIRST EVIDENCE OF GLAUCOMA	CHAMBER ANGLE	MEANS USED TO RELIEVE GLAUCOMA
20. C. S.	Extracapsular	4 days; hyphemia 5th day	3 months	Trabecular adhesions in lower temporal area; obliterated elsewhere	Filtering cicatrix?
21. J. S.	Intracapsular	5 days	5 weeks	Obliterated	Filtering bleb
22. T. D.	Extracapsular	1 day	1 month	Open 12 : 30 and 6 o'clock; obliterated elsewhere	Miotics
23. J. G.	Extracapsular	4 days	1 month	Obliterated	Operation
24. L. H.	Extracapsular	7 days; prolapse flap 5th week	1 month	Obliterated	Operation (loosening of prolapse)
25. M. K.	Extracapsular	3 days	6 months	Trabecular adhesions 5 to 7 o'clock; obliterated elsewhere	Uncontrolled
26. S. M.	Extracapsular	?	2 months	Obliterated	Operation
27. R. B.	Extracapsular	7 days	3 weeks	Open 11 to 12 o'clock	Normal with eserine (usually)
28. B. C. (R) . .	Extracapsular	3 days	4 months	Obliterated	Operation
29. B. C. (L) . .	Loop	?	1 month	Obliterated	Operation
30. A. M.	Loop, intracapsular	?	7 months	Obliterated	Operation
31. J. L.	Extracapsular	2 days	1 month	Obliterated	Operation
32. E. B.	Extracapsular	Iris incarcerated	7 weeks	Obliterated	Uncontrolled
33. C. C.	Extracapsular	7 days	5 months	Obliterated	Operation
34. H. D.	Extracapsular	12 days	3 months	Obliterated	Filtering cicatrix?; miotics
35. N. E.	Intracapsular	7 days; hyphemia	7 months	Obliterated	Operation
36. H. F.	Extracapsular	10 days	1 month	Obliterated	Operation
37. L. H.	Extracapsular	10 days	1 month	Practically obliterated	Operation
38. J. W.	Extracapsular	13 days	1 month	Obliterated	Operation
39. V. D.	Extracapsular	12 days	3 months	Obliterated	Tension 37.5 once; filtering cicatrix?
40. E. R.	Extracapsular	12 days	6 weeks	Obliterated	Operation

extending at least as far as the one third of the trabeculum nearest the cornea, and usually as far as the end of Descemet's membrane (Table IX). In the two exceptions, in which only small areas remained

open, the tension returned to normal with miotics. It must be remembered that the anterior third of the trabeculum does not usually contain trabecular spaces (Fig. 295).



FIG. 295. Angle in patient with secondary glaucoma following cataract extraction, showing complete peripheral anterior synechia. The arrows point to the end of Descemet's membrane where the iris is adherent.

In the group in which the glaucoma was first noted after discission of an after-cataract (12 eyes), none gave evidence that increased tension had been present previous to the discission operation (Table X). Since this operation often results in collapse of the anterior chamber and is usually associated with at least a mild degree of congestion of the ciliary body, it is reasonable to assume that discission may be the precipitating factor in this type of glaucoma.

Postoperative collapse of the anterior chamber cannot be held entirely responsible for formation of synechias in both groups of cases. Additional factors in their formation may be the injudicious postoperative overuse of atropine sulfate and the organization of repeated anterior chamber hemorrhages.

When one finds a fairly large area of open angle in an eye with glaucoma following removal of the crystalline lens, it is probable that the cause falls into one of the four following categories: (1) glaucoma capsulare; (2) active or healed iridocyclitis or cyclitis; (3) probable simple glaucoma in which the cataract operation was merely coincidental; and (4) epithelial ingrowth.

TABLE X

GLAUCOMA FOLLOWING DISCISSION OF AFTER-CATARACT

PATIENT	TYPE OF CATARACT OPERATION	INTERVAL BETWEEN OPERATION AND FORMATION OF ANTERIOR CHAMBER	INTERVAL BETWEEN CATARACT OPERATION AND DISCISSION	INTERVAL BETWEEN DISCISSION AND FIRST EVIDENCE OF GLAUCOMA	CHAMBER ANGLE	MEANS USED TO RELIEVE GLAUCOMA
1. T. L.	Extracapsular	3 weeks	11 weeks	5 months	Obliterated	Operation
2. C. C.	Extracapsular	1 day (hy- phemia 6th day)	7 weeks	8 weeks	Nearly obliterated; anterior part of trabeculum visible	Operation
3. M. K.	Extracapsular	5 days	6 weeks	2 days	Trabecular adhe- sions 2 to 8 o'clock; obliter- ated elsewhere	Miotics
4. J. B.	Loop; extracapsular	2 days	3 months	2 months	Obliterated	Operation
5. L. C.	Extracapsular	?	5 weeks	6 weeks	Operation
6. A. F.	Extracapsular	4 days	10 weeks	10 weeks	Obliterated	Miotics (poor physical condi- tion)
7. A. S.	Extracapsular	?	3 months	6 weeks	Trabecular adhe- sions 3 and 8 o'clock; obliter- ated elsewhere	Miotics
8. E. J.	Extracapsular	?	10 weeks	5 months	Trabecular adhe- sions 6 to 8 and 2 to 4 o'clock; ob- literated elsewhere	Operation
9. W. M.	Extracapsular	3 days	5 weeks	19 months	Obliterated	Operation
10. A. P.	Extracapsular	10 days	5 months	8 months	Operation
11. A. S.	Extracapsular	?	2 months	2 weeks	Obliterated	Operation
12. C. L.	Extracapsular	6 days	7 weeks	11 months	Trabecular adhe- sions below; obliterated above	Miotics

Occasionally one finds a case of surgical aphakia with a completely closed chamber angle and either no evidence of glaucoma or only an occasional rise in tension. In several cases of this type, I found that incarcerated iris tissue was present in the wound. A filtering bleb or cicatrix was probably the cause of the absence of glaucoma. Cases 8, 14, 15, 20, 34, and 39 in Table IX were of this type. In case 21 a definite filtering bleb was present. In one other case of

postoperative aphakia with a closed angle the tension never was found to be over 28 mm. of mercury (Schiotz), although the patient was examined daily for two months after a conjunctival flap was drawn over the area of the incision. In this case the chamber had formed on the second postoperative day and was collapsed on the fourth day. It remained flat for twenty-six days and reformed only after the flap was drawn. Choroidal separation was visible while the chamber was flat. The original chamber collapse as well as the persistence of normal tension was undoubtedly due to a minute fistula.

That closure of the chamber angle may, in itself, give rise to glaucoma in otherwise normal eyes is highly significant from the point of view of human physiology. It emphasizes the view that Schlemm's canal is the chief structure concerned in resorption of water from the eye.

One means of preventing the formation of peripheral synechias by facilitating the formation of the anterior chamber, is the use of a corneoscleral suture at the time of operation. Statistical results published by Leech and Sugar and by McLean, showed a decrease in the number of cases of delayed formation of the anterior chamber when corneoscleral sutures were used as compared with the cases in which conjunctival sutures were used. The sutures used in the cases reported by Leech and Sugar included both modified Liégard and Verhoeff sutures. McLean employed a suture which he introduced at the Wilmer Institute. Another means of preventing synechia formation, the injection of air into the chamber, has been advocated by MacMillan, McLean, and others.

Cyclodialysis has been accepted by most ophthalmic surgeons as the treatment of choice for glaucoma which follows operations for cataract. It is the only operation which can be safely done in aphakic eyes. The operative site may be anywhere along the circumference of the angle except at the region of the iridectomy and the limbs of the coloboma. These latter places are avoided to prevent undue trauma or hemorrhage. A total of 56 cyclodialyses were performed on the series of 52 eyes in which glaucoma had occurred following

cataract operations. Forty-one per cent were successful, and 59 per cent unsuccessful. Further consideration will be given to this operation in the next section.

*Alterations Following Operations on Narrow-Angle Glaucoma
(Acute Glaucoma and "Chronic Congestive Glaucoma")*

Iridectomy. Since von Graefe's introduction of iridectomy as a means of relieving glaucoma, many theories have been advanced to explain its action, particularly in acute glaucoma. Weber expressed the belief that the operation reopens the closed chamber angle. Czermak asserted that cutting the angle tissues facilitates direct communication between the anterior chamber and Schlemm's canal. De Wecker believed that a filtering scar, with or without incorporation of uveal tissue, results from iridectomy. Priestley Smith found a filtering cicatrix after iridectomy histologically in one case. Troncoso, with gonioscopic evidence, stated that reopening the closed angle is not necessary for the success of iridectomy for acute glaucoma. He supported the idea that the operation works by "re-establishment of free communication between the anterior and posterior segments of the eye."

My observations of 13 cases of acute glaucoma in which the tension was reduced to normal after the use of miotics (Table VIII), indicate that the reduction in tension was associated with the opening of at least a portion of the circumference of the closed angle. It, therefore, seems logical that iridectomy must function in the same way, that is, by freeing a sufficiently large portion of the angle circumference to permit normal function of the trabecular area and Schlemm's canal and at the same time prevent further contact between the iris and trabecular wall. However, actual examination of 14 iridectomized eyes in which the tension became normal, showed that 8 had partial opening of the angle and 6 had completely closed angles. Of those with partly open angles, 2 had been subjected to true basal iridectomies, while in the other 6 the iris was plastered against the trabecular wall. In 5 of the 6 eyes with completely closed angles filtering cicatrices were believed to account for the normalization

of tension. Small, ragged pieces of iris tissue could be seen in the operative incision and sometimes even projecting out from under the conjunctiva. Filtering blebs were present in 2 of the 5. In the others, pressure on the globe through the eyelids produced small areas of conjunctival edema. The sixth case was not included since the lens had been removed after the iridectomy so that interpretation of the action of the iridectomy might be equivocal. In 5 of the 6 cases in which the angles were closed and the tension was normal, the reduction of tension following application for two minutes of the new Schiötz tonometer with the 15.5 gm. weight was measured and compared with the tension in a series of 26 control cases. In the 5 patients the tension dropped 16 to 7.5 mm. In the normal controls it dropped 2.5 to 6.5 mm. In 2 successfully iridectomized patients with partly open angles it dropped only 3 mm. of mercury in each case, indicating absence of external filtration.

Six other eyes with acute glaucoma were examined after unsuccessful iridectomy. All had completely closed angles. At the operative area the iris root was plastered against the trabeculum.

These findings support the belief of Weber and of de Wecker and are probably best expressed by the statement of Fuchs that "iridectomy acts primarily by opening the closed chamber angle, a thing which is possible only when the angle has not been permanently closed by exudation. After permanent closure by a peripheral synechia, iridectomy can do good only in case it produces a filtering cicatrix."

A study of the relation between success of the iridectomy and the length of time between the onset of the attack of acute glaucoma and the time of iridectomy indicated an inverse relationship between the two (Table XI). If operation was done before dense adhesions had formed, part of the closed angle was opened and the tension reduced. If the adhesions had become too firm, the operation was unsuccessful except when a filtering cicatrix resulted.

A study of these results leads to the conclusion that iridectomy alone is the procedure of choice for acute glaucoma during the interval between attacks, or after relief from an attack has been ob-

TABLE XI

RELATION BETWEEN THE INTERVAL BEFORE IRIDECTOMY
AND THE END RESULTS OF OPERATION

PATIENT	INTERVAL BETWEEN ONSET OF ATTACK AND IRIDECTOMY	SUCCESS OR FAILURE IN REDUCING TENSION TO NORMAL	ANGLE AFTER OPERATION
1. L. E.	2 months	Failure	Not seen
2. E. A. (right eye)	2 weeks	Failure	Not seen
3. A. J. (right eye)	3 weeks	Failure	Not seen
4. A. J. (left eye)	3 weeks	Failure	Not seen
5. R. H. (right eye)	3 weeks	Failure	Not seen
6. M. P.	11 days	Failure	Not seen
7. M. S.	9 days	Failure	Obliterated
8. K. K.	10 days	Success	Partly open
9. L. S.	4 days	Success	Partly open
10. E. A. (left eye)	2 days	Success	Not seen
11. F. B.	2 months	Success; filter- ing cicatrix	Obliterated
12. M. T.	2 months	Success; filter- ing cicatrix	Obliterated
13. A. M.	5 weeks	Success; filter- ing cicatrix	Obliterated
14. C. E.	2½ weeks	Success; filter- ing cicatrix	Obliterated
15. J. S.	3 weeks	Success	Partly open (basal iridectomy)
16. R. M.	2 weeks	Success; gross filtering cicatrix	Obliterated
17. A. K.	10 days	Success; gross filtering cicatrix	Obliterated

tained with miotics. Early in an attack it would also serve to break the vicious combination of progressive angle blockage and increasing tension and would tend to reopen the angle. Late in an attack, however, after firm adhesions have formed, perhaps a combination of iridectomy with partial inclusion of the iris might well be sub-

stituted for the classic operation. The *ab externo* approach might also tend to insure the incarceration of tags of iris in the wound and thus promote filtration.



FIG. 296. Drawing of angle showing cyclodialysis. (Courtesy of Otto Barkan.)

Cyclodialysis. When Heine in 1905 introduced cyclodialysis as a surgical method of relieving glaucoma, he expressed the belief that a communication between the anterior chamber and the suprachoroidea became the channel of drainage. Others, Salus among them, maintained that the procedure acted by severing afferent arteries and injuring nerves, thereby causing partial atrophy of the ciliary body and subsequently decreased formation of aqueous. No histologic evidence was submitted until Elschnig's report in 1932 of an eye on which a successful cyclodialysis had been performed

fourteen years previously. He found an opening between the anterior chamber and the supraciliary space in the area of operation.

The first gonioscopic study of cyclodialysis was made by Vannas,



FIG. 297. Successful cyclodialysis. Note the adhesion between the two portions of the open cleft.

in 1935. He studied 37 cases and found patent clefts in 20 and none in 13. The findings were uncertain in 4. He reported normal tension in 30 of the 37. Four of the eyes with open clefts had been operated on from eight to thirteen years previously.

In 1936, Barkan, Boyle, and Maisler reported the gonioscopic data in 14 cases of simple glaucoma in which cyclodialysis had been done. In 11 in which the operation had been successful, a patent cleft was found. In 3 cases in which it had been unsuccessful, reattachment of the ciliary body had occurred. In 2 of the latter a second cyclodialysis was successful and resulted in the formation of a patent supraciliary cleft.

In a study of 65 cyclodialyses on eyes with narrow-angle glaucoma or with glaucoma simplex, I found an "all or none" relation between success of the operation and the formation of a communication between the anterior chamber and the supraciliary space. If a cleft is present the tension is reduced to normal; if it is not, the tension remains uncontrolled (Figs. 296, 297). Of the 65 cyclodialyses, 34 were successful in reducing the tension to normal. In 33 of the 34 there were sizable patent clefts. In these the tension varied from 8 to 21 mm. of mercury (Schiotz), averaging 15 mm. In the remaining cyclodialysis the tension at times reached

32 mm. of mercury and required miotics for control. In this case there was a very small patent cleft extending about 10 degrees along the angle circumference. There was no apparent relation

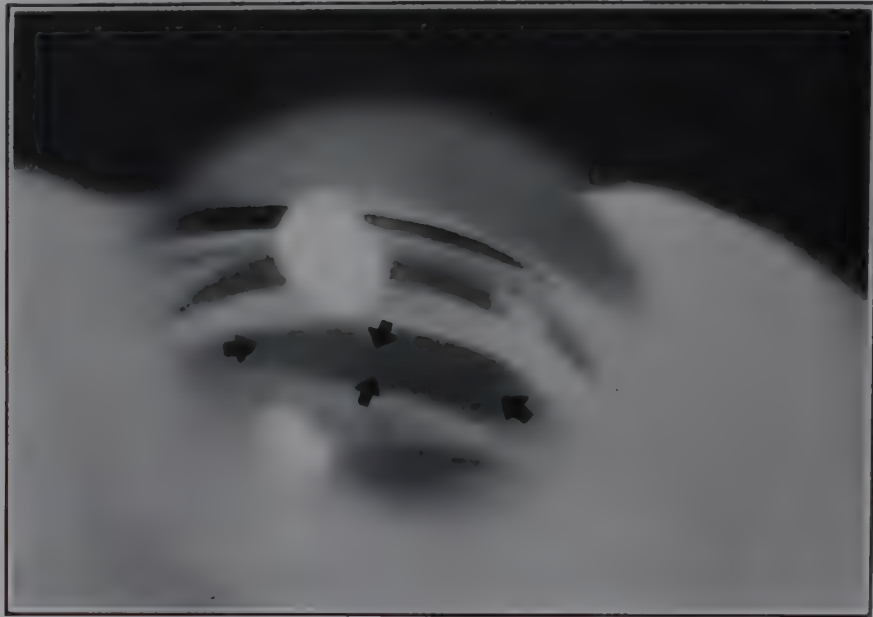


FIG. 298. Widely open cyclodialysis. Arrows point to cleft.

between the tension and the breadth of the cleft in the 33 cases with sizable cyclodialysis openings.

Of the 31 unsuccessful cyclodialyses, none showed patent clefts. In nearly all there was stripping of a portion of Descemet's membrane; and in some, reattachment of the ciliary body posterior to its original position. Occasionally, after a cyclodialysis operation, one sees what appears superficially to be an open cleft; but closer examination, usually aided by transillumination of the sclera at the area of operation, reveals that it really ends in a blind pocket after extending only a short distance. Such cases indicate that the "all or none" law does not always agree with the gonioscopic picture in those instances where the cleft is shallow. In some cases, where the cleft is very broad, the lamina fusca of the sclera becomes visible (Figs. 298, 299, 300).

Similar evidence of the "all or none" relation between success of cyclodialysis and the presence of a patent supraciliary cleft was seen in the series of 56 cyclodialyses performed on eyes with glaucoma following cataract operations.

Occasionally normalization of the tension following cyclodialysis

is found to be due to another agent than an open cleft. In one eye in which the tension remained normal in spite of absence of any visible cleft, examination revealed that subconjunctival filtration



FIG. 299. Widely open cyclodialysis. Arrows point to cleft.

was taking place through a scleral fistula at the site of the scleral incision. The spatula had penetrated into the vitreous and an incarceration of choroid in the scleral wound had resulted in fistulization (Fig. 301).

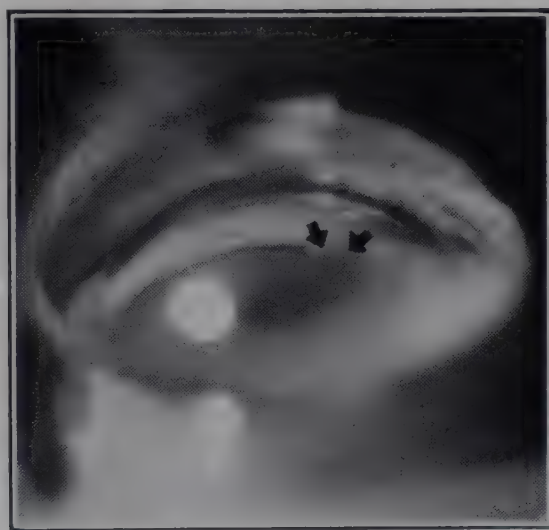


FIG. 300. Small open cyclodialysis. Arrows point to cleft.

The presence of peripheral anterior synechias in the chamber angle before operation has little effect on the success of cyclodialysis. Twenty-four of the 65 cyclodialyses for narrow-angle and simple glaucoma were observed preoperatively. Seven had completely closed angles, and 4, angles that were partly open. Function-

ing cyclodialysis clefts resulted in 5 of the 7 eyes with completely closed angles and in 2 of the 4 with partly open angles.

Cyclodialysis is usually not advocated for narrow-angle glaucoma



FIG. 301. Filtering choroidal incarceration at the site of the cyclodialysis incision in an eye with a closed cyclodialysis cleft.

but rather for chronic simple glaucoma, for glaucoma following surgery for cataract, and for instances of glaucoma due to posterior subluxation of a clear lens. However, in my series the results in the narrow-angle cases do not differ as markedly from the others as one might expect. The operation was successful in 3 out of 8 eyes with acute glaucoma, in 3 out of 5 with "chronic congestive glaucoma," in 27 out of 52 with glaucoma simplex, and in 23 out of 56 with glaucoma following cataract operations. It is true that in the narrow-angle types of glaucoma, congestive episodes may occur which result in closure of the clefts. Of the 6 successful cases of cyclodialysis in narrow-angle glaucoma, a review after another interval of five to six years revealed that 2 were normal and had open clefts, 1 experienced an acute attack nine months after operation, 1 had an acute attack after four years but was normalized and retained an open cleft after treatment with miotics, 1 failed after five years when surgery for cataract was done, and the remaining 1 could not be followed. A

similar effect is produced when cataract removal becomes necessary after a successful cyclodialysis. The postoperative reaction may close the cleft, and repetition of the cyclodialysis may become necessary. However, in 2 instances the cyclodialysis cleft remained open in spite of subsequent surgery for cataract.

A follow-up study of cyclodialyses for simple glaucoma and for glaucoma following cataract surgery in a group of patients who were first studied up to five years after operation and reviewed after another period of five to six years, indicated that closure of previously-open clefts may take place also in a small number of these cases. Of 28 successful cyclodialyses for simple glaucoma, 11 remained normal and had open clefts, 13 could not be followed, 2 remained normal until cataract surgery was done, 1 returned after an interval of five years with a hard eye and 1 continued to have low tension but the cyclodialysis could not be seen. Of the 23 successful cyclodialyses for glaucoma following cataract surgery, 7 remained normal and had open clefts, 10 could not be followed, and 6 became failures or partial failures although in 4 of these, small, open, but shallow, partially functioning clefts were present.

The "all or none" relation between success of the cyclodialysis operation and the presence of a supraciliary cleft makes possible the gonioscopic determination of the prognosis even though the tension remains normal for a considerable interval of time following operation. This interval was determined in 19 eyes with simple glaucoma in which the clefts had closed and the tension had become elevated above 28 mm. of mercury (Schiotz). In 9 instances the tension was elevated on the patient's first postoperative visit. In 2 instances each there were normal tensions for three, five, and six weeks, respectively. In 1 each there were normal tensions for one, four, nine, and sixteen weeks, respectively. In other words, except for those eyes in which the tension was elevated at the time of the first postoperative examination, the interval between unsuccessful operation and recurrence of ocular hypertension varied from one to sixteen weeks, averaging five and eight-tenths weeks. Thus the absence of any cleft a week or two after operation indicates that the operation

has failed in spite of the fact that the tension may be low at the time of gonioscopic examination.

The cause of failure of many well-performed cyclodialyses is undoubtedly the persistence and organization of a blood clot in the cleft. I have observed the process of such closure in 2 instances. A review of the hospital records of the 121 cyclodialyses discussed above to determine the relation between the incidence of hyphemia and success of the operation revealed that hyphemia occurred in 52.9 per cent of the successful "primary" cases and 70.9 per cent of the unsuccessful ones. In the cases of secondary glaucoma following cataract surgery it occurred in 60.8 per cent of the successful and 81.8 per cent of the unsuccessful ones. It is thus apparent that hyphemia does not preclude success but it is also true that lack of success is associated with a greater incidence of hyphemia. In the total of 121 cyclodialyses, hyphemia occurred in 56.1 per cent of the successful, and 76.5 per cent of the unsuccessful cases. To avoid this complication, to minimize trauma, and to avoid pain, several suggestions may be made. The Blaskovics modification of the cyclodialysis procedure, the avoidance of the horizontal meridian where the long posterior ciliary vessels and nerves (as well as nerve loops) are located, and the use of eserine salicylate postoperatively to keep the cleft open, may help. After use of a smaller spatula sweep, I believe that a dialysis involving 120 degrees of angle circumference is better. For previously unoperated eyes, the operation may be done in the upper half of the angle and the patient should be propped up in bed after operation to keep hyphemia out of the cleft. If the eye has previously been operated upon, the dialysis is done below. The patient should lie on one side following operation. Air injection into the anterior chamber after cyclodialysis, especially in aphakes, will probably improve upon the relatively poor results with this operation.

Elliot Trephining Operation. Trephining operations (Fig. 302), like all fistulizing procedures, depend on the subconjunctival drainage of intra-ocular fluid. The patency of the new channel of outflow chiefly determines the success of the operation. Other factors which must be considered in relation to success of trephining are the

presence of preoperative synechias, the formation of postoperative synechias, and the type of glaucoma affecting the eyes treated with this operation.

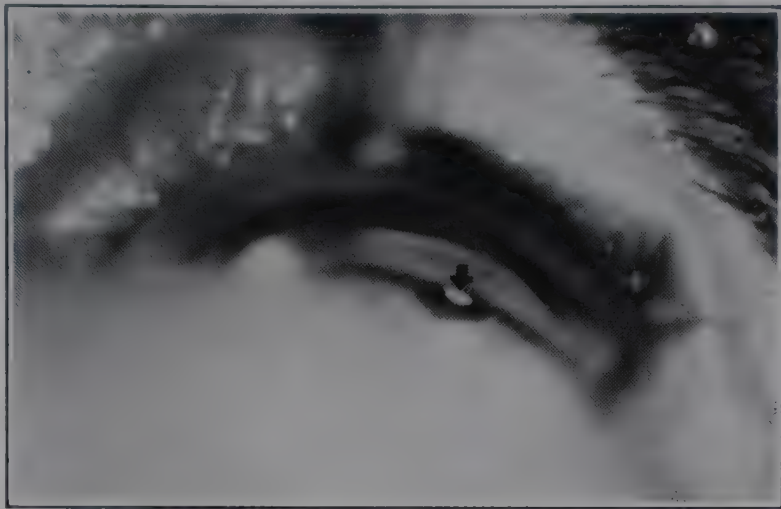


FIG. 302. Open trephine (indicated by arrow). The dark area below this is the pigment of the ciliary body seen through the peripheral iridectomy.

Obstruction of the trephine hole by iris, ciliary body, or lens results in failure of the operation (Figs. 303, 304). Less obvious causes of failure are scarring of the conjunctival bleb and the presence of



FIG. 303. Partially filled trephine opening. Portions of the iris pillars and one ciliary process are adherent to the walls of the trephine opening.

a connective tissue membrane across the outer scleral aperture. These causes of failure usually result from postoperative inflammation. Troncoso and Reese, in 1933, reported the gonioscopic data on 29 trephined eyes. They found adhesions to the sclera in 7, incarceration of the ciliary processes in 12, and exudates attached to

the ciliary body and sclera in 6. Seven had consecutive iritis and only 3 showed normal healing. In a series of 86 trephinings in which the trephine holes were visible gonioscopically, I found 52 with normal

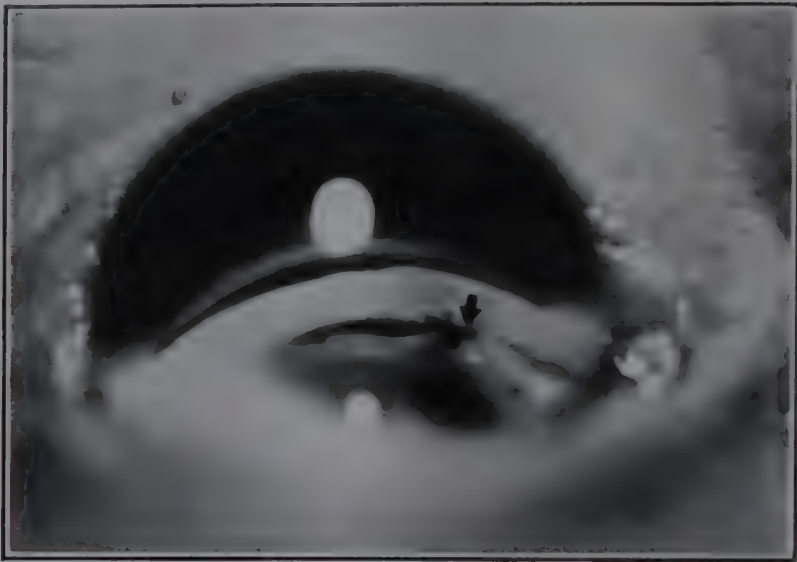


FIG. 304. Trephine plugged by iris.

tensions; the tension in 12 was controlled with miotics and that in 22 was uncontrolled. In 15 of the 52 eyes with normal tension the trephine hole was partly plugged by iris, ciliary body, lens or sclera. In 10 of the 12 in which the tension was controlled with miotics, the openings were partly obstructed. In addition one opening contained a connective tissue membrane. In 20 of the 22 uncontrolled cases the openings were completely plugged by iris, ciliary body, or lens. In the remaining 2 the trephine holes were patent but

TABLE XII

RELATION BETWEEN TENSION CONTROL AND PATENCY OF TREPHINE OPENINGS

TENSION	VISIBILITY OF TREPHINE OPENING		VISIBLE TREPHINE OPENINGS WITH PARTIAL OR COMPLETE PLUGGING
	NOT SEEN	SEEN	
Normal, 101	49	52	15 partly filled
Controlled with miotics, 22	10	12	10 partly filled
Uncontrolled, 31	9	22	20 completely plugged
Total, 154	68	86	45

were covered with scarred, nonfiltering conjunctiva. These statistics (Table XII) appear to justify the conclusion that the success of trephining depends on the efficacy of subconjunctival drainage at the site of the trephine hole.

In a considerable number of eyes — both those which had been operated on successfully and those which had not — trephine openings were not visible gonioscopically. Among the causes of this obscuration of the trephine openings are placing the trephine opening too far back, adhesions between the anterior lens capsule and the anterior lip of the trephine, and extremely shallow chamber angles. The adhesions to the anterior lip of the trephine obviously occur during the postoperative period while the anterior chamber is collapsed.

The presence of synechias in the chamber angle preoperatively affects the operative results somewhat because of the fact that the iris root no longer is at its normal position on the ciliary body but is adherent further forward on the trabeculum or on the corneal margin. Consequently, there is greater danger of the trephine opening being plugged by ciliary processes or by iris. Preoperative and postoperative study of the angles in 64 trephined eyes was made to determine the relation between preoperative synechias and the success of the trephining operation (Table XIII). The angles were open preoperatively in 52 eyes, partly open in 7 and completely closed in 5. The tension was reduced to normal in 35 of the 52 with open angles, in 4 of the 7 with partly open angles, and in 2 of the 5 with closed angles. The same series of eyes were studied to determine the relation of the operation to the formation of synechias postoperatively. Of the 52 angles which were open preoperatively, 2 remained open, 34 remained partly open, and 16 closed completely. Of the 7 angles which had been partly open preoperatively, 5 remained unchanged, and 2 became completely closed. These results corroborate previous evidence that trephining operations have a great tendency to cause the formation of peripheral anterior synechias. The same statement is true for all operative procedures

TABLE XIII

RELATION BETWEEN SYNECHIAS, TENSION CONTROL, AND
PATENCY OF TREPHINE OPENINGS IN SIXTY-FOUR
TREPHINED EYES SEEN GONIOSCOPICALLY BOTH
PREOPERATIVELY AND POSTOPERATIVELY

TYPE OF GLAUCOMA	ANGLE BEFORE OPERATION	TENSION	ANGLE AFTER OPERATION	PATENCY OF TRE- PHINE OPENING	
"Acute congestive," 6	Open, 3	Normal, 2	Obliterated, 2	Patent, 1 Not seen, 1	
	Partly open, 1	Uncontrolled, 1 Controlled with miotics, 1	Obliterated, 1 Partly open, 1	Plugged by iris, 1 Patent, 1	
	Obliterated, 2	Normal, 1 Uncontrolled, 1	Obliterated, 1 Obliterated, 1	Patent, 1 Not seen, 1	
"Chronic conges- tive," 3	Open, 2	Normal, 2	Partly open, 1	Iris adherent to anterior lip, 1	
	Partly open, 1	Normal, 1	Obliterated, 1 Obliterated, 1	Not seen, 1 Not seen, 1	
Chronic simple, 54	Open, 47	Normal, 31	Open, 1	Patent, 1	
			Partly open, 21	Patent, 14 (3 partly filled with ciliary processes, 4 with iris, 1 with lens)	
				Not seen, 6	
				Iris adherent to anterior lip, 1	
			Obliterated, 9	Patent, 5 (1 partly filled with iris, 1 with sclera)	
				Not seen, 4	
			Controlled with miotics, 7	Partly open, 6	Patent, 4 (3 partly filled with iris, 1 with lens)
				Not seen, 2	
			Obliterated, 1	Patent, 1 (partly filled with iris)	
			Uncontrolled, 9	Open, 1	Patent, 1
		Partly open, 6		Patent, 1; bleb scarred	
				Plugged, 5 (1 filled with ciliary proc- esses, 4 with iris)	
		Obliterated, 2		Plugged, 1 (filled with ciliary processes)	
				Not seen, 1 (too far back)	
Partly open, 5	Normal, 3	Partly open, 2	Patent, 1 (partly filled with iris)		
			Not seen, 1		
	Uncontrolled, 2	Obliterated, 1 Partly open, 2	Patent, 1 Plugged, 2 (filled with iris, ciliary process)		
Obliterated, 2	Controlled with miotics, 1	Obliterated, 1	Not seen, 1		
	Uncontrolled, 1	Obliterated, 1	Not seen, 1		
Absolute glaucoma (chronic simple), 1	Obliterated, 1	Normal, 1	Obliterated, 1	Patent, 1	

which are followed by collapse of the anterior chamber for any considerable length of time.

The relation of the presence of postoperative synechias to the success of the trephining operation was first pointed out by Werner, who, from a study of 14 trephined eyes, concluded that synechias which form after trephining have no significance so far as the action of the operation in reducing ocular hypertension is concerned, since the outflow is no longer dependent on natural channels. I studied a group of 154 cases of trephining, postoperatively, and similarly found that there is no parallelism between the presence of postoperative synechias and the success or failure of trephining. In the 154 cases of trephining (Table XIV), the operation was successful in 101, required the additional use of miotics in 22, and was unsuccessful in 31. In the first group, the angle was open in 7 per cent, partly open in 48 per cent, and entirely closed in 46 per cent. In the group of 22 trephinings in which the tension was normalized with miotics, 1 had open angles, 16 had partly open angles, and 5 entirely closed angles. Of the 31 unsuccessful trephinings, the angle was open in 1, partly open in 15, and entirely closed in 15. Although these statistics indicate that the presence of postoperative synechias are not ordinarily of importance when the trephine opening is patent, they are important when the trephine opening is completely or nearly completely plugged. Then the drainage from the eye is further embarrassed by the newly formed synechias and the ocular tension may rise above its preoperative level.

The aforementioned group of 154 trephinings was subdivided according to the types of glaucoma present, to determine whether trephining is equally successful in all of the types. Of the 154, 15 were done on eyes with acute glaucoma, 16 on eyes with "chronic congestive glaucoma," 18 on eyes with simple glaucoma, and 5 on eyes with absolute glaucoma (simplex). The tension was reduced to normal in 9 of the eyes with acute glaucoma, in 9 of those with "chronic congestive glaucoma," in 12 of those with simple glaucoma, and in 3 of those with absolute glaucoma. Obviously the operation may be equally effective in all these types of glaucoma.

TABLE XIV

RELATION BETWEEN SYNECHIAS, TENSION CONTROL, AND
PATENCY OF TREPHINE OPENINGS IN A TOTAL
OF ONE HUNDRED AND FIFTY-FOUR TREPHINED
EYES EXAMINED POSTOPERATIVELY

TYPE OF GLAUCOMA	TENSION CONTROL	ANGLE AFTER OPERATION	PATENCY OF TREPHINE OPENING
"Acute congestive," 15	Normal, 9	Partly open, 3 Obliterated, 6	Not seen, 3 Patent, 3 Not seen, 3
	Controlled with miotics, 1 Uncontrolled, 5	Partly open, 1 Obliterated, 5	Patent, 1 Plugged, 3 (1 filled with ciliary body and iris, 1 with iris, 1 with lens) Not seen, 2
"Chronic congestive," 16	Normal, 10	Partly open, 4	Patent, 1 Not seen, 2 Iris adherent to anterior lip, 1
		Obliterated, 6	Patent, 2 Not seen, 3 Late infection, 1
	Controlled with miotics, 3	Partly open, 3	Patent, 1 (partly filled with ciliary process) Not seen, 2
Chronic simple, 118	Uncontrolled, 3	Partly open, 2	Plugged, 2 (1 filled with iris and ciliary process, 1 with iris) Not seen, 1
	Normal 79	Obliterated, 1	Patent, 2
		Open, 7	Not seen, 3 Iris adherent to anterior lip, 2
		Partly open, 39	Patent, 23 (4 partly filled with ciliary processes, 5 with iris, 1 with lens) Not seen, 13 Iris adherent to anterior lip, 3
		Obliterated, 33	Patent, 20 (1 partly filled with iris and ciliary process, 1 with iris, 5 with ciliary processes, 1 with sclera, 1 with lens) Not seen, 11 Iris adherent to anterior lip, 1 Lens capsule adherent to anterior lip, 1 Connective tissue in trephine, 1
	Controlled with miotics, 17	Open, 1	Patent, 7 (4 partly filled with iris, 1 with ciliary process, 1 with lens) Not seen, 5
		Partly open, 12	Patent, 2 (partly filled with iris) Not seen, 1 Ciliary process adherent to anterior lip, 1
	Uncontrolled, 22	Obliterated, 4	Patent; bleb scarred Patent, 1; bleb scarred
		Open, 1 partly open, 13	Plugged, 11 (2 filled with iris and ciliary processes, 8 with iris, 1 with ciliary processes) Not seen, 1

TABLE XIV *Continued*

TYPE OF GLAUCOMA	TENSION CONTROL	ANGLE AFTER OPERATION	PATENCY OF TREPHINE OPENING
Absolute glaucoma (Chronic simple), 5		Obliterated, 8	Plugged, 3 (filled with ciliary processes) Not seen, 4 Late infections, 1
	Normal, 3	Partly open, 2 Obliterated, 1	Not seen, 2 Patent, 1
	Controlled with miotics, 1	Obliterated, 1	Not seen, 1
	Uncontrolled 1	Obliterated, 1	Plugged by lens, 1

Improper placing of the trephine opening is an important contributing factor to the failure of trephining. Proper placement requires knowledge of the varying amounts of scleral overlap around the corneal circumference. LaGrange's figures of 1.75 mm. of overlap above, 1.45 below, and 1.0 mm. at the sides indicate that a trephine opening properly placed above would be too far back if made at the same position on either side. Similarly, if the angle is closed preoperatively, the trephine may enter the posterior chamber behind the area of iris adhesion and permit easy access of the ciliary processes to the trephine opening. Rapid release of aqueous when the bulging iris is opened at the time of operation may be one of the important factors in causing prolapse of ciliary processes into the hole.

LaGrange Sclerectomy. Anatomically this operation has the same effect as trephining. Only four operations of this type were studied. One was successful, two required the addition of miotics for control, and one remained uncontrolled. In the successful one, performed on a patient with acute glaucoma, a patent trephine-like opening was visible gonioscopically. In two of the remainder, incarcerations of ciliary processes were present in the sclerectomy opening.

Iris Inclusion Operations (Iridotaxis, Iridencleisis). Like the trephining and sclerectomy operations, the iris inclusion operations depend on the subconjunctival drainage of aqueous. Unlike the

former, however, the outflow channels are not usually visible gonioscopically. The iris incarcerations can always be seen but their functional status has to be determined from the clinical findings



FIG. 305. Incarceration of the iris in a case of successful iridencleisis. The arrows point to the keratome incision.

(Fig. 305). It appears that normal function is most often associated with the inclusion of a thick layer of iris tissue in one edge of the wound rather than with a thin, atrophic layer stretched along the entire length of the scleral incision. Probably the pressure of the two lips of the incision on the latter hinders filtration, while if the iris is jammed into one corner, there is a tendency for the lips to separate.

In addition to the functional efficacy of the new outflow channels, the possibility of interference with the old natural channels must be considered. The presence of peripheral anterior synechias preoperatively does not apparently have much effect on the outcome of iris inclusion operations. In a preoperative and postoperative study of 13 such operations, 9 had open angles preoperatively, 3 were partly open, and 1 completely closed. Of the 13 preoperatively open angles, the tension was reduced to normal by operation in 6, was controlled with miotics in 1, and remained uncontrolled in 2. In the eyes with preoperatively partly open angles and in the eye with the closed angle the operations were all successful.

The postoperative formation of peripheral anterior synechias influences the operative results only when the new drainage channel is nonfunctioning. Then, of course, the added embarrassment of

peripheral anterior synechias decreases the amount of natural drainage to an even lower level than before operation. A study of 48 iris inclusion operations postoperatively was made to determine the relation between operative success and the presence of postoperative synechias, without considering the functional status of the subconjunctival drainage channel (Table XV). In 33 cases the ten-

TABLE XV

RELATION BETWEEN SYNECHIAS AND TENSION CONTROL
IN FORTY-EIGHT OPERATIONS FOR INCLUSION OF
THE IRIS

TYPE OF GLAUCOMA	TENSION	ANGLE BEFORE OPERATION	ANGLE AFTER OPERATION
"Acute congestive," 5	Normal, 1	Partly open, 1
	Controlled with miotics, 1	Obliterated, 1
	Uncontrolled, 3	Obliterated, 3
"Chronic congestive," 8	Normal, 6	Open, 1	Obliterated, 2
			Partly open, 4
	Controlled with miotics, 2	Open, 1	Partly open, 1
Chronic simple, 34	Normal, 25	Open, 4	Partly open, 20
		Partly open, 2	
		Open, 1	Obliterated, 5
		Obliterated, 1	
	Controlled with miotics, 3	Partly open, 3
Absolute (chronic simple), 1	Uncontrolled, 6	Open, 2	Partly open, 5
			Obliterated, 1
	Normal, 1	Partly open, 1	Partly open, 1
	Totals	13	48

sion was reduced to normal; in 6 it was controlled with the addition of miotics, and in 9 it was uncontrolled. Of the normalized cases the angles were partly open in 26 and closed in 7.02. Of those controlled with the addition of miotics, 4 were partly open and

2 closed. And of the 9 uncontrolled cases, 5 were partly open and 4 closed. No parallelism between success or failure of the operations and the presence of postoperative synechias is apparent.

To prevent the lips of the scleral incision from squeezing the iris between them and thus hindering the exit of aqueous to the subconjunctival tissues, the use of a scleral punch forceps to make a small sclerectomy of the anterior lip is helpful.

Goniotomy. Barkan, in 1936, advocated a new operation called goniotomy or goniotrabeculotomy for the relief of certain cases of glaucoma simplex. He believed that incision of the inner wall of Schlemm's canal by producing a direct opening between the anterior chamber and the canal, would relieve the glaucoma, although this is not in accord with our present knowledge of the physiology of the angle. I reported a series of 9 cases (including 6 cases of glaucoma capsulare in which capsular exfoliations have the same effect that pigment does in Barkan's cases) in which goniotomy or actual curettage of the trabeculum was performed, and concluded that the operation may be successful if the incision severs the scleral spur and allows fluid from the anterior chamber to enter the supraciliary space, as in cyclodialysis. At any rate, in the series described, the operation had only a temporary effect on the ocular tension. Further reports by Barkan on the use of goniotomy in cases of hydrophthalmos indicate that the operation may be quite effective in these cases, where it probably acts to restore access of aqueous directly to the trabecular wall by removing obstructing persistent fetal tissue from a portion of the angle circumference.

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INDEX TO VOLUME 1

INDEX TO VOLUME I

- Abbé microscope, 28, 32
 Seidentopf modification, 28
 Aberration of lenses, 7, 23
 Abiotrophy, 159
 Abrasions, of cornea, 542
 Acarus demodex folliculorum, 265
 Accommodation, depth of anterior chamber in, 613
 oblique illumination of, 1, 2
 Acne rosacea conjunctivitis, 228
 keratitis, 229, 476
 direct focal illumination of, 481
 narrow beam in, 477
 optic section in, 477
 sclerotic scatter in, 481
 retro-illumination of, 477
 Addison's disease, 169
 melanin pigmentation in, 168
 Adrenalin, effect of, on conjunctival circulation, 134
 Afocal illumination of corneal nerves, 277
 Age, conjunctiva in, 159
 cornea farinata in, 342
 cornea, infiltration of, in, 449
 eyelid margins in, 259
 lens, zones of discontinuity in, 103
 linea corneae senilis in, 362, 381
 marginal ulcer of cornea in, 529
 melanin pigment in, 370
 Mooren's ulcer in, 529
 senile degenerations in, 302
 senile marginal atrophy in, 363, 365
 Stähli-Hudson line in, 362, 381
 Allergic congestion, of eyelid borders, 260
 Allergic conjunctivitis, 189
 Allergy, in eye diseases, 189
 Anatomy, of anterior chamber, 558
 of chamber angle, 618
 of conjunctiva, 124
 of cornea, 274
 Anesthesia, corneal, in leprosy, 518
 Angioma, of conjunctiva, 248
 Aniline dyes, and vital staining of conjunctiva, 148
 Aniridia, 621
 Ankyloblepharon, in burns of conjunctiva, 230
 Anomalies, of anterior chamber, congenital, 568
 of conjunctiva, 154
 absence of, 154
 of arrangement, 155
 congenital tumors, 154, 155
 cryptophthalmia, 154
 developmental, 154
 partial metaplasia, 154
 pigmentation, 154
 of cornea, developmental, 290
 buphthalmos, 295
 cornea plana, 297
 embryotoxon, 301
 keratoconus posticus, 297
 megalocornea, 291
 microcornea, 290
 opacities, congenital, of, 298
 of posterior corneal surface, 299
 zonular of, 298
 posterior crater-like formations, 297
 Anterior chamber, 558
 anatomy of, 558
 angle of, 618
 acuity of, 609
 anatomy of, 618
 congenital alterations in, 627
 aniridia, 630
 biomicroscopy of, 596, 598
 Koeppel method, 597
 congenital alterations in, 627
 cysts of, 632
 examination of, 66, 599
 foreign bodies in, 615, 642
 glaucoma and, 630
 following cataract extraction, 645
 gonioscopy of, 596, 599
 in megalocornea, 291, 629
 hydrophthalmos of, 627

Anterior chamber—(*Continued*)angle of—(*Continued*)

- inflammatory alterations in, 633
- in iris, congenital coloboma of, 620
- in megalocornea, 291, 620
- pathology of, 621
- photography of, 606
- postoperative alterations in, 633
- traumatic alterations in, 631
- tumors of, 632

anomalies of, congenital, 568

aqueous changes in, 588

flare, 560

aqueous of, turbidity of, 588

biomicroscopy of, 559

in buphthalmos, 568

caterpillar hairs in, 587

cholesterol in, 593

cilia in, 586

colloidometer, Roenne, 560

contusions of, 577, 585

crystalline deposits in, 593

cysticercus cellulosae in, 581

cysts of, 569, 580

epithelial downgrowth, 581

implantation, 580

depth of, 120, 558, 609

abnormalities of, 568

in accommodation, 613

anatomic factors affecting, 611

errors of refraction, 611

growth of lens, 612

size of eye, 613

in ciliary body congestion, 614

in contusion, 577

in errors of refraction, 611

growth of lens, 612

Hartinger formula, 120

measurement of, with Ulbrich micrometer, 119, 559

by parallelepiped, 559

pathology in, 595

physiologic factors in, 613

accommodation, 613

ciliary body congestion, 614

pupillary dilatations, 614

in pupillary dilatation, 614

size of eye and, 613

diffuse illumination of, 564

direct focal illumination of, 564

epithelial downgrowth in, 581

examination of, 559

Anterior chamber—(*Continued*)

foreign bodies in, 586

glass membranes in, 578

in glaucoma, operations for, 651

gonioscopy of, 596, 598, 599, 619

hemorrhage in, 373, 577

deposits on cornea in, 439, 577

hyaline membranes in, 572

hyphemia in, 577

blood staining of cornea in, 373

following iridectomy for shallow-

angle glaucoma, 651

iris coloboma, 570

keratic precipitates in, 426

in megalocornea, 568

membranes in, 569

in microcornea, 569

in microphthalmos, 569

with narrow beam, examination of,

595

neoplastic growths near angle of,

593

optic section of, 563

parasites in, 581

parallelepiped, corneal, 564

pathology of, 587, 595

pigmented bodies in, 569

pupillary membrane in, 569

retro-illumination of, 564

scintillatio nivea, in, 594

shallow, 595

specular reflection in, 564

synechiae, congenital anterior, in, 572

thermal convection currents in, 565

in trauma of eyeball, 572

Tyndallimetry of, 561

vitreous herniations into, 585

prolapse into, 581

Anterior microphthalmos, 290

Apparatus, description of, 14

Aqueous, changes in, 588

flare, 588

use of hand slit lamp in, 58

opacities in, 589

in iritis, 593

turbidity of, in, 588

corpuscular elements in, 588

diffuse, 588

Aqueous fluid, floating cells in, 11

Aqueous solutions, in biomicroscopy,
286, 287

Arc lamp, 15

Arc lamp—(*Continued*)

- anterior chamber, examination of, with, 559
- intensity of, 24, 25
- lens for, 23
- Stähli-Hudson line, examination of, with, 381
- Arcus juvenilis, 303
- Arcus lipoides, 302
- Arcus pinguiculus, 302
- Arcus senilis, 302
 - optic section of, 303
- Argyrosis, of conjunctiva, 174
 - of cornea, 387
 - optic section of, 391
 - parallelepiped in, 391
 - retro-illumination of, 391
 - endogenous, 387
- Ariboflavinosis, 401
- Arruga screw, 24
- Arteriosclerosis, conjunctival capillaries in, 133
- Aspergillus fumigatus, in cornea, 531
- Asteroid hyalitis, 594
- Atrophy, senile marginal, of cornea, 363
- Aubert binocular corneal microscope, 2
- Aubert-Czapski binocular corneal microscope, 2, 4
- Avitaminosis A, effect of, on conjunctiva, 167
 - in xerosis corneae, 483
- Axis, of beam, 2
 - of illumination, 66
 - of microscope, 66
 - of observation, 2, 66
- Azur ii, vital staining with, of conjunctiva, 149
 - of cornea with, 286
 - of herpes simplex corneae, 463
 - of herpes zoster keratitis, 466
 - of orifices of meibomian glands, 257
 - of phlycten, 482
 - of pinguecula, 165
- Bandform keratitis, 317, 343
 - etiology of, 343
 - optic section of, 346
 - retro-illumination of, 346
 - sclerotic scatter in, 346
- Bausch and Lomb hand slit lamp, 59
- Beam, astigmatic, in studying corneal thickness, 444

Beam—(*Continued*)

- cylindrical, in examination of anterior chamber contents, 564
- focal part of, 2
 - for illumination of retina, 5
 - rectangular, 6
- focused, history of, 5
- of light, 65
 - narrow, 78
 - in keratic precipitates, 427
 - luminous, optical characteristics of, 24
- Bedewing, of cornea, 146, 394
 - of corneal epithelium, 285
 - endothelial, 442
 - retro-illumination of, 442
 - epithelial, 146
 - dystrophy, 318
 - in herpes simplex corneae, 462
 - retro-illumination of, 285
 - in ruptures of Descemet's membrane, 362
- Bengal rose, vital staining with, of conjunctiva, 137
- Berger loupe, 5
- Biber lattice-like dystrophy, 335
- Binocular loupe, 5
- Binocular microscope, 52
 - Czapski, 29
 - corneal, Czapski, 28
 - Greenough type, 37
 - stereoscopic visualization in, 69
- Biomicroscope, adjustment of, 65
 - Arruga screw for, 24
 - Aubert, 2
 - Comberg, 35
 - condensing system of, 20
 - description of, 14
 - diaphragm of, 20
 - Elschnig screw for, 24
 - Fincham, 53
 - focusing of, 67
 - Goldmann (Haag-Striet), 56
 - Gullstrand, 7, 14
 - Haag-Striet, 56
 - hand slit lamp, 58
 - Henker, 8, 14
 - history of, 1
 - Cherubra d'Orleans, 29
 - illuminating lens of, 22
 - system of, 14
- illumination, 69. (*See also* Illumination)

Biomicroscope—(*Continued*)

- Koeppé silvered mirror for, 50
- tube for, 21
- Köhler system of illumination for, 8
- Lemoine and Valois, 53
- lens, rectangular, for, 24
- light sources of, 52
- Mayou, 56
- oculars for, 31
- magnifications of, 31
- Poser, 41
- illuminating system of, 47
- stereopsis, in use of, 68
- Universal, simplified model, 38
- paired objectives, magnifications of, in, 41, 44
- Universal-Koeppé, 37

Biomicroscopy, of abrasions of cornea, 542

- of acne rosacea keratitis, 477
- of acute conjunctivitis, 185
- of allergic congestion of eyelid borders, 260
- of anterior chamber, 559
- angle of, 66, 596
- cysts in, implantation, 580
- of arcus juvenilis, 303
- of arcus senilis, 303
- of aqueous fluid, 11
- of argyrosis, 174
- of bandformed opacity of cornea, 344
- of Bitot's spots, 166. (*See also* Conjunctiva)
- of caterpillar hairs in anterior chamber, 587
- of conjunctiva, 134
- degenerative changes in, 159
- injuries of, 229
- lymphatics of, 148
- normal, 137
- papilloma of, 231
- pinguecula of, 161
- vessels of, 138, 140
- of conjunctivitis, 176, 185, 189
- of cornea, 274
- abrasions of, 542
- changes in, 392, 447
- congenital zonular opacity of, 298
- foreign bodies in, 553
- inflammations of, 447
- nerves of, 277, 442

Biomicroscopy—(*Continued*)

- of cornea—(*Continued*)
- normal, 274
- plana, 297
- scars of, 531
- tuberculosis of, 508
- ulcer of, 520, 522
- Mooren's, 529
- vascularization, superficial, of, 403
- of cysts, implantation, 580
- of Descemet's membrane, folds in, 421
- development of, 1
- of developmental anomalies of conjunctiva, 154
- of cornea, 290
- diminution in transparency, of, 290
- of dystrophy, macular, 335
- of episcleritis, 270
- of epithelioma, 237
- of eyelashes, 257
- of eyelid margins, 252
- of parasitic infestations of, 265
- of follicular conjunctivitis, 194
- of foreign bodies, conjunctival, 230
- corneal, 553
- of fornix, 139
- of fundus, 66
- of Gaucher's disease, 165
- and gonioscopy, 598
- of granulomatous tumors, 251
- of herpes simplex corneae, 462
- history of, 1
- illumination, focal, in, 9
- of injuries, thermal, chemical, and radiational, 555
- of iris, neoplasms of, 593
- of Kayser-Fleischer ring, 386
- of keratic precipitates, 440
- of keratitis, 447. (*See also* Keratitis)
- filamentary, 490
- interstitial, 492
- avascular, 503
- e lagophthalmos, 471
- leprous, 515
- neuroparalytic, 489
- parenchymatous, 492
- infiltrative, 515
- profunda, 518
- superficial punctate, 450
- of keratoconjunctivitis sicca, 470

Biomicroscopy—(*Continued*)

- of keratoconus, 353
- of Krukenberg's spindle, 372
- of lacrimal caruncle, 147
- of lacrimal puncta, 265
- of lattice-like dystrophy, 335
- lens, plastic, in, 600
- of lipin keratitis in Hurler's syndrome, 351
- localization in, 111
 - by direct focal illumination, 111
 - by focusing microscope, 117
 - in neovascularization of cornea, 408
 - by optic section, 111
 - by parallactic displacement, 122
- of malignant nevus of conjunctiva, 243
- of megalocornea, 291
- of neovascularization of conjunctiva, 177
- of neuromparalytic keratitis, 488, 537
- of nodular dystrophy of cornea, 325
- of pannus trachomatosa, 210
- of papillary hypertrophy of conjunctiva, 178
- of parenchyma, changes in, 414
- in Parinaud's oculoglandular syndrome, 220
- of pemphigus of conjunctiva, 222
- of pericorneal injection, 402
- of phlyctenular conjunctivitis, 190
- of pigmentation, superficial vortical, 371
- of pinguecula, 161
 - hypertrophied, in Gaucher's disease, 165
- of plica semilunaris, 147
- procedure for, 122
- of pterygium, 305
- of scleritis, 271
- technique of, 64
 - adjustment of instrument, 65
 - contact glass, 597
 - dark adaptation of examiner, 65
 - diffuse illumination, 69
 - direct focal illumination, 72
 - eyeball, survey of, 64
 - illumination, 69
 - diffuse, 69
 - direct focal, 72

Biomicroscopy—(*Continued*)

- technique of—(*Continued*)
- illumination—(*Continued*)
 - indirect, 105
 - proximal, 105
 - of room, 64
- Koeppel method, of examining anterior chamber, 597
- localization, 111
- luminous fixation targets in, 65
- narrow beam in, 78
- optic section in, 111
- oscillatory field in, 108
- parallelepiped in, 74
- patient, management of, for, 65
- precautions, 65
- procedure for, 122
- projection of shadows in, 108
- retro-illumination in, 79, 91
- sclerotic scatter in, 70
- specular reflection in, 97
 - examination by, 91
- stereopsis in, 68
- Ulbrich micrometer, 119
- vital staining and, 137, 148, 286
- terminology of, 11
- of thermal convection currents, 565
- of trachoma, 201
- of vernal conjunctivitis, 192
- of vitreous humor, 66
- of vitreous herniations in anterior chamber, 585
- Birth injuries, of cornea, 422, 424
 - glass membranes in, 578
- Bishop-Harman hand slit lamp, 62
- Bitot's spots, of conjunctiva, 160, 166, 487
- Bitumi microscope, 32
- Blebs, of conjunctiva, 222
 - of cornea, in optic section, 109
 - of lens, in optic section, 109
 - retro-illumination of, 446
 - in thinning of cornea, 446
- Blennorrhoea, 200
 - inclusion, 200
- Blepharitis, 260
 - chronic, 260
 - madarosis in, 260
 - trichiasis in, 260
- Blood staining, of cornea, 373
- Bouquets, of conjunctiva, 185
- Bowen's disease, 237

- Bowman's membrane, changes in, 409
 folds in, 105
 parallelepiped in, 410
 shagreen in, 342
 tears in, 412
- Bowman's zone, changes in, 409, 410
 folds in, 410
 honeycomb effect in, 411
 opacities in, 410
 tears in, 412
 vascularization of, 413
- Brawny scleritis, 271
- Bulbar conjunctiva, 140
- Bullae, in direct focal illumination, 109
 epithelial, of cornea, 395, 399
 in diffuse vesicular keratitis, 488
 in optic section, 399
 parallelepiped of, 400
 retro-illumination of, 399
- Buphthalmos, 291, 295
 deepening of anterior chamber in, 568
 optic section in, 296
 rupture of Descemet's membrane in, 296
 tears, in Bowman's membrane in, 412
 in Descemet's membrane in, 422
- Burns, of conjunctiva, 230, 555
 of cornea, 555
 acid, 555
 alkali, 555
 chemical, 555
- Calcareous deposits, in conjunctiva, 160
- Canal, Schlemm's, 623
- Canaliculitis, 269
- Canaliculus, lacrimal, 265
- Carbon arc lamp, 16
- Carcinoma, squamous-cell, of eyelid, 265
- Caruncle, lacrimal, 147
- Cataract, focal illumination of, 74
 oblique illumination of, 2
 retro-illumination of, 79
 "sunflower," in Kayser-Fleischer ring, 386
 surgery of, Descemet's membrane in, 421
 fluorescent ultraviolet light in, 18
 glaucoma following, 645
 biomicroscopy in, 581
- Catarrh, vernal, 192
 corneal changes in, 449
- Catarrhal conjunctivitis, 223, 449
- Caterpillar hairs, in anterior chamber, 587
 conjunctivitis due to, 227
 in cornea, 228
- Catoptric images, 92
 specular reflection, zone of, 92
- Caustic surface of object, 7
- Chalazion, 261
 optic section in, 261
- Chalcosis, of cornea, 391
- Chamber, anterior. *See* Anterior chamber.
- posterior. *See* Posterior chamber.
- Chancre, of conjunctiva, 215
- Chemical injuries, of conjunctiva, 230
 of cornea, 555
- Chemosis, of conjunctiva, 176, 557
- Cherubra d'Orleans, 29
- Cholesterol, in anterior chamber, 593
- Christian-Schüller's disease, 165
- Cicatrix, cystoid, 548
- Cilia, 257
 loss of, 210, 260
- Ciliary body, congestion of, and depth
 of anterior chamber, 614
- Coats' white rings, of cornea, 340
- Cocaine anesthetic, as vasoconstrictor, 134
 in vital staining, 149, 287
 of epithelium, 287
- Colloidometer, Roenne, 560, 588
- Comberg biomicroscope, 35
- Condensing system, 20
- Congenital anomalies, of anterior chamber, 568
 of angle, 627
 of cornea, 298
- Congenital syphilis, 492
- Congenital tumors of conjunctiva, 155
- Congestion, of bulbar conjunctiva, 185
 of ciliary body, 614
- Conical cornea, 353. (*See also* Keratoconus)
- Conjunctiva, 124
 in acne rosacea, 228
 in Addison's disease, 169
 angioma of, 248
 anomalies, developmental, of, 154
 of arrangement, 154, 155
 congenital tumors, 154, 155
 melanosis oculi, 155
 pigmentation, 154

Conjunctiva—(*Continued*)

argyrosis of, 174
 in arteriosclerosis, 133
 biomicroscopy of, 134
 Bitot's spots in, 160, 166, 487
 vitamin A deficiency in, 167
 blood supply of, 126
 bulbar, 140
 optic section of, 140
 vessels in, 140
 burns of, 230
 calcareous deposits in, 160
 capillaries of, 133
 caterpillar hairs in, 227
 chemosis of, 176
 circulation of, effect of drugs on, 134
 vasoconstrictors, 134
 vasodilators, 134
 cold light lamp for examining, 137
 cysticercus in, 236
 cysts of, 235
 narrow beam in, 235
 degeneration, fatty, of, 165
 lipoid, of, 165
 degenerative changes of, 159
 senile, 159
 calcareous deposits in, 160
 diaphanoscopy of, 136
 diffuse illumination of, 135
 direct focal illumination of, 135
 diseases of, 176
 edema of, 176
 epithelial layer of, 37
 optic section of, 137
 epithelioma of, 236
 epithelium of, 125
 in erythema multiforme (*exudativa*),
 223
 in erythema nodosum, 223
 fatty degeneration of, 165
 filaria loa in, 236
 film line in, 137
 follicles on, 139, 183
 follicular conjunctivitis, 194
 foreign bodies in, 230
 of fornix, 139
 granuloma of, 251
 gumma of, 219
 hemorrhages of, 229
 in herpes zoster ophthalmicus, 200
 histology, normal, of, 124
 in hydroa vacciniform, 227

Conjunctiva—(*Continued*)

hyperemia of, 176
 illumination of, 135
 yellow filter, 137
 inclusion blennorrhea, 200
 indirect illumination of, 136
 inflammations of, 176. (*See also* Con-
 junctivitis)
 injection of, 176
 injuries of, 229. (*See also* Injuries)
 cysts, due to, 235
 keratoconjunctivitis, epidemic, 195
 lacrimal caruncle, in disease of, 147
 leprosy of, 219
 limbus of, 145
 lipoid degeneration of, 165
 lupus of, 214, 509
 lymphatics in, 148
 malignant tumors of, 231, 243
 melanosis of, 243
 neovascularization of, 177
 nerves of, vital staining of, 149
 nevus of, 242
 normal, biomicroscopy of, 137
 histology of, 124
 in ochronosis, 169
 ophthalmia nodosa of, 227
 optic section of, 135
 palpebral, biomicroscopy of, 137
 papillae of, 178
 examination of, 139
 in Parinaud's syndrome, 220
 in partial metaplasia, 154
 pemphigus of, 222
 pigmentation of, 168. (*See also* Pig-
 mentation)
 developmental, 154
 pinguecula of, 161
 congenital, 155
 plica semilunaris and, 147
 polyps of, 251
 proximal illumination of, 136
 pterygium in, 305
 red free light, examination of, with,
 137
 scars of, 230
 siderosis of, 175
 specular reflection of, 136
 subepithelial layer of, 126
 biomicroscopy of, 138
 superficial layer of, 125
 symblepharon in, 222

Conjunctiva—(*Continued*)

- syphilis of, 215. (*See also* Syphilis)
- tarsal, biomicroscopy of, 137
 - epithelium of, 125
- trachoma, follicles in, 183. (*See also* Trachoma)
- tubercle of, 213. (*See also* Tubercle)
- tuberculides of, 214
- tuberculoma of, 214
- tuberculosis of, 213
- in tularemia, oculoglandular, 220
- tumors of, 231. (*See also* Tumors)
 - congenital, 155
- in varicella, 468
- vascular buds in, 139
- vascularization of, 126
- vessels of, 138
 - biomicroscopy of, 140
 - bulbar, 126
 - circulation of, 132
 - tarsal, 126
- vital staining of, 148
 - anesthesia in, 148
 - aniline dyes in, 148
 - azur ii in, 149
 - bengal rose in, 137
 - cresyl blue in, 149
 - of epithelium, 137
 - fluorescein in, 149
 - method of, 148
 - methylene blue in, 149
- in vitiligo, 173
- wounds of, 230
- xanthomatous patches in, 165
- xerosis of, 166, 210

Conjunctival vessels, biomicroscopy of, 138

Conjunctivitis, 176

- acne rosacea and, 228
- acute, 185
- allergic, 189
- catarrhal, 223
 - direct focal illumination of, 449
- caterpillar hairs in, 227
- chronic, 189
- diagnosis of, 176
- follicles in, 183
- follicular, 194
 - differentiation of, from trachoma, 135
 - optic section in, 195
 - in Parinaud's oculoglandular syndrome, 221

Conjunctivitis—(*Continued*)

- granular, 215
 - hyperemia in, 176
 - inclusion blennorrhea, 200
 - Leishmania, 518
 - leprosy in, 219. (*See also* Leprosy)
 - neonatorum, 200
 - neovascularization in, 177
 - nodosa, 227
 - papillae in, 178
 - papulosa, 215
 - Parinaud's, 220. (*See also* Parinaud's syndrome)
 - phlyctenular, 185, 190, 481
 - biomicroscopy of, 190
 - simple, direct focal illumination of, 449
 - spring, 192
 - subacute, 189
 - swimming-bath, 200
 - syphilitic, 215
 - trichomatous. *See* Trachoma.
 - traumatic, 229
 - tuberculous, 213
 - vernal, 192
 - abortive type, 194
 - bulbar type, 193
 - optic section in, 193
 - palpebral type, 192
 - viral, 195
 - virus inclusion, 200
- Contact glass, examination of anterior chamber with, 596
- Contact lens for examination, of anterior chamber, 600
- of deep vitreous and retina, 50
- Contact lenses, gonioscopic, 600
- application of, 605
- Contusions of cornea, 541
- narrow beam in, 585
- Copper deposits in cornea, 391
- Cornea, 274
- abrasion of, vital staining of, 288, 542
 - optic section in, 543
 - acne rosacea in, 228, 476
 - anatomy of, 274
 - anomalies of, 290
 - anterior surface of, optic section of, 278
 - parallelepiped of, 278
 - aqueous solutions, in vital staining of, 287
 - arcus juvenilis in, 303

Cornea— (*Continued*)

- arcus pingvicularis in, 302
- arcus senilis in, 302
- argyrosis of, 387
- aribo flavinosis, effect of, on, 401
- aspergillus fumigatus in, 531
- azur ii, vital staining of, with, 286, 288
- bedewing of, 285, 318
- biomicroscopy of, 274, 392
- blebs of, in optic section, 109
- blood staining of, 373
 - optic section in, 377
- Bowman's membrane of, changes in, 409
 - folds in, 411
 - honeycomb effect in, 411
 - opacities of, 410
 - optic section in, 410
 - tears of, 412
 - vascularization of, 413
- burns of, 555
 - chemical, 555
 - radiational, 557
 - superficial, 557
 - thermal, 555
- calcareous film in, 343
- caterpillar hairs in, 227
- cells of, vital staining of, 288
- chalcosis of, 391
- cicatricial processes of, 531
- congenital anomalies of, 299
 - hyaline membrane, 299
 - opacities of, 298
 - posterior corneal surface, 299
 - zonular, 298
- conical, 353
- contusion of, 541
- copper deposits in, 391
- curvature of, abnormalities in, 297
 - cornea plana, 297
 - keratoconus posticus, 297, 363
 - posterior crater-like formations, 297
- cysts, retrocorneal, 424
- degeneration of, band-shaped, 343
 - calcareous, 368
 - crystalline parenchymatous, 352
 - deep crocodile shagreen, 342
 - Groenouw's nodular familial, 332
 - hyaline, 368
 - marginal, 363
 - pigmentary, 370
 - senile, 302

Cornea— (*Continued*)

- degeneration of— (*Continued*)
 - superficial, folds in Descemet's membrane, 417
- Dellen of, 306
- deposits in, retro-illumination of, 91
- dermoid tumors of, 155
- Descemet's membrane, changes in, 409, 656. (*See also* Descemet's membrane)
- Descemet's zone, 415. (*See also* Descemet's zone)
- diffuse illumination of, 274
- direct focal illumination of, 73, 74. (*See also* Direct focal illumination)
- in dystrophia adiposa, 316
- dystrophy of, 307. (*See also* Dystrophy)
 - Fuchs', 309, 317
- ectasia of, 363, 532
- edema of, 393
 - retro-illumination of, 285
- embryotoxon in, 291, 297, 301, 316
- epithelial bullae of, 399
 - changes in, 400
 - mucus, 108
- epithelioma of, 236
 - intra-epithelial of, 237
- epithelium of, 281
 - bedewing of, 285, 309
 - droplets in edema of, 119
 - vital staining of, 287
- erosions of, 543
 - multiple epithelial, 457
 - optic section in, 457
- examination of, biomicroscopic, 274
- face of, posterior, 279
- facets of, 306
- farinata, 316, 342
 - parallelepiped in, 342
 - retro-illumination of, 342
- fatty degeneration of, 538
- Fleischer's ring of, 362
- fluorescein, vital staining of, with, 289
- fluorescence of, 18
- foreign bodies in, 548, 553
 - glass, 555
 - gold, 554
 - metallic, 554
 - optic section of, 553
 - projection of shadows in, 108
- Fuchs' dimples in, 306

Cornea—(*Continued*)

- fungus infections of, 531
- gerontoxon in, 302
- glass membranes of, specular reflection of, 105
- globosa, 291
- Groenouw's nodular dystrophy of, 324
- guttata, 319, 320
 - indirect retro-illumination of, 321
 - parallelepiped in, 320
 - retro-illumination of, 311, 321, 322
- Haab's striae in, 296
- Hassall-Henle bodies of, 302, 305
 - of Descemet's membrane, 279
- herpes of, 461
 - keratitis metaherpetica, 467
- honeycomb effect in, 411
- Hudson's line in, 381
- hyaline membranes of, specular reflection of, 105
 - on posterior corneal surface, 299
- in hydroa vacciniforme, 227
- illumination of, 392
- infiltration of, 449
 - in acne rosacea keratitis, 476
- inflammation of, 447. (*See also* Keratitis)
 - superficial zone, 448
- injection of, pericorneal, 402
- injury of, 424, 541, 547
 - chemical, 555
 - contusions, 541
 - optic section of, 424
 - radiational, 557
 - thermal, 555
 - traumatic, 541
- insect stings of, 557
- internal specularity of, 276
 - in optic section, 285
- iron deposits in, 391
- Kayser-Fleischer ring in, 385
- keratic precipitates on, 119, 426
- keratitis, 447. (*See also* Keratitis)
- keratoconjunctivitis, sicca, 470
 - epidemic, 195
- keratoconus, 353. (*See also* Keratoconus)
 - posticus, 297, 363
- keratomalacia, 483
- keratomycosis in, 531
- Krukenberg's spindle in, 372
- lacerations of, 550

Cornea—(*Continued*)

- leprosy of, 515
- leukoma of, congenital, 299
 - indirect illumination of, 107
- limbal spur, 285
- lime burns of, 556
- linea senilis in, 362, 381
- liquid precorneal layer, 279
- localization of, 111. (*See also* Localization)
 - with hand slit lamp, 58
 - optic section in, 111
 - measurement of distance of pathologic features, 116
- lupus of, 509
- macula of, 532
- marginal dystrophy of, 363
 - oblique illumination of, 365
- megalocornea, 291
- mercurochrome in, vital staining of, 286
- methylene blue, in vital staining of, 286, 288
- microcornea, 290
- micrometry, 120
- mucus on, vital staining of, 287
- mustard gas burns of, 556
- nebula of, 532
 - projection of shadows in, 108
- neofornation of vessels of, 402
- nerves of, 277, 442
 - nerve fibers, increased visibility of, 362
 - optic section in, 277
 - retro-illumination of, 518
 - sclerotic scatter of, 518
 - vital staining of, 443
 - with methylene blue in limbal region, 289
- nevus of, 242
- nile blue sulphate, in vital staining of, 288
- nodular dystrophy, Groenouw's, of, 324, 325
- noninflammatory changes of, 307
- normal, 274
 - diffuse illumination of, 274
 - parallelepiped of, 288
- opacities of, 316
 - bandformed, 343
 - circular peripheral, 340
 - in keratoconus, optic section of, 361

Cornea—(Continued)

opacities of—(Continued)

ribbon-shaped, 343

zonular, 343

optic properties of, 274

optic section of, 279

of anterior surface of cornea, 278

osmic acid solution, in vital staining
of, 289

pannus of, 403

parallelepiped of, 74

anterior surface, 278

parenchyma of, dystrophy of, 311

staining of cells in, 288

parenchymatous keratitis, 492

pemphigus of, 223

pericorneal injection of, 402

pigment deposits on, 435

pigmentation of, 302, 369. (*See also*
Pigmentation)

plana, 297

polychrome methylene blue, in vital
staining of, 286, 288posterior crater-like formations of,
297

posterior surface of, 279

retro-illumination of, 91

precorneal film of, 276

importance of, 281

origin of, 279

vital staining of, 281, 287

pterygium of, 305

punctate dots in, 400

relucency of, 274

retro-illumination of, 81

sclerotic scatter in, 71

relucant superficial limbal spur in,
285

reparative processes of, 415

in interstitial keratitis, 501

reticular degeneration of Biber, Dim-
mer, and Haab, 335retro-illumination of, 79, 83, 89. (*See*
also Retro-illumination)

rodent ulcer of, 529

rose bengal, in vital staining of, 286,
288

salmon patch of, 408

scars of, 531

diffuse illumination of, 70, 110

direct focal illumination of, 74

narrow beam in, 532

Cornea—(Continued)

scars of—(Continued)

optic section in, 112

parallelepiped of, 532

sclerotic scatter in, 532, 548

thinning of, in optic section, 445

sclerotic scatter of, 70

shagreen of, 342

siderosis of, 391

silver deposits in, 387

size, changes in, 290

sodium fluorescein, in vital staining
of, 286

specular reflection of, 91, 94

specularity of, internal, 276

Stähli-Hudson line in, 381

stings of, insect, 557

stroma of, alterations in, 413

clefts in, 413

fissures, 413

stroma of, focal illumination of, 276.
(*See also* Focal illumination)

infiltration of, 414

narrow beam of, 78. (*See also* Nar-
row beam)

scar formation in, 415

vascularization of, 415

surfaces of, specular reflection of, 97

surgical incisions of, 551

syphilis of, 503

thickness of, 116, 120

changes in, 445

increase in, 444

optic section of, 443, 444

thinning of, 445

in keratoconus, 355

optic section of, 355, 445

in trachoma, 211

transparency of, 290

parallelepiped of, 290

tuberculosis of, 508, 509

tumors of, nevus, 242

papilloma, 231

Turk's line in, 371

ulcer of, 520

atheromatous, 530

catarrhal, 529

dendritic, 463

keratomycosis, 531

marginal, 525

sclerotic scatter of, 529

Mooren's, 529

Cornea—(*Continued*)

- ulcer of—(*Continued*)
 - Morax-Axenfeld, 522
 - optic section of, 523
 - ulceration of, erythema multiforme, 223
 - ulcus serpens of, 523
 - vacuoles of, optic section of, 109
 - in varicella, 468
 - vascularization of, 401, 413
 - pericorneal injection, 402
 - deep, 403
 - vernal catarrh involving, 449
 - verticillium graphii in, 531
 - vessels of, 408
 - deep, in optic section, 409
 - parallelepiped of, 409
 - neoformation of, 402
 - retro-illumination of, 408, 409
 - viruses in, 468
 - vital staining of, 286
 - vortex dystrophy of, 371
 - war gases, effect of, on, 556
 - white limbus girdle in, 366
 - white rings in, optic section of, 340
 - whorl opacity of, 371
 - wounds of, 547
 - optic section of, 549
 - perforating, 550
 - specular reflection of, 550
 - zones of specular reflection in examination of, 91, 94
- Corneal block in retro-illumination, 79
- Corneal epithelium, 285
- Corneal microscope, Aubert, 2
 - Aubert-Czapski, 2, 4
 - history of, 4
- Corneal nerve fibers, direct focal illumination of, 362
- Corneal nerves, increased visibility of, 442
 - diffuse illumination of, 70
 - vital staining of, 443
- Corneal precipitates, retro-illumination of, 86
- Corneal stroma, direct focal illumination of, 103
 - dystrophy of, 316. (*See also* Dystrophy)
- Cornu cutaneum, 265
- Cresyl blue, in vital staining, of conjunctiva, 149

Cresyl blue—(*Continued*)

- in vital staining—(*Continued*)
 - of lattice-like dystrophy, 338
 - of lymphatics, 148
- Cryptophthalmia, 154
- Cyclodialysis, for glaucoma following cataract extraction, 650
 - gonioscopy of, 654
- Cysticercus cellulosae, 581
 - of conjunctiva, 236
- Cysts, of anterior chamber, 569, 580
 - of conjunctiva, 235
 - cysticercus, 236
 - filaria loa, 236
 - retention cysts, 235
 - of cornea, retrocorneal, 424
 - implantation, 580
- Czapski corneal binocular microscope, 8, 28
- Dark adaptation, 65
 - field illumination, 83
- Defects, congenital, of anterior chamber, 568
 - of conjunctiva, 155
 - of cornea, 298
 - developmental, of conjunctiva, 154
 - of cornea, 290
- Degeneration, calcareous, of cornea, 368
 - fatty, 165
 - of cornea, 538
 - hyaline, of cornea, 368
 - lipin, 538
 - lipoid, 165
 - marginal, of cornea, 363
- Degenerative changes, of conjunctiva, 159
- Dellen, 306
- Deposits, calcareous, in conjunctiva, 160
 - crystalline, in anterior chamber, 593
- Dermoid tumors, of bulbar conjunctiva, 155
 - of cornea, 155
 - optic section of, 155
- Dermolipoma, 155, 159
- Descemetitis, 426
- Descemetocoele, 446
- Descemet's membrane, argyrosis of, 387
 - fold of, 105, 414, 417
 - in corneal edema, 414
 - diffuse illumination of, 70, 421
 - specular reflection in, 103

- Descemet's membrane— (*Continued*)
 Hassall-Henle bodies in, 279, 305
 laceration of, glass membranes in, 580
 ruptures of, 542
 in buphthalmos, 296
 in keratoconus, 360
 retro-illumination of, 361
 specular reflection of, 103
 tears in, 422
 differentiation of, from folds, 105
 specular reflection of, 103
 trauma of, 580
 warts in, 305
 specular reflection of, 103
- Descemet's zone, alterations in, 415
 endothelium in, edema of, 415
 inflammation of, 492
 hyaline excrescences in, 416
 striae, 416
 inflammation of, 492
 optic section of, 415
- Developmental anomalies. *See* Anomalies.
- of conjunctiva, 154
 of cornea, 290
- de Wecker ophthalmomicroscope, 3
- Diaphanoscopy, 79
 of conjunctiva, 136
- Diaphragm, 20
 double slit, 20
 disk, circular revolving, 20
 López-Lacarrère, 20
 Poser biomicroscope, 50
 rectangular aperture, 50
- Diffuse illumination, 69
 of conjunctiva, 135
 of cornea, normal, 274
 of corneal nerves, 70
 of corneal scars, 70, 110, 537
 of Descemet's membrane, folds of, 70, 421
 of Fuchs' dimples, 306
 of Kayser-Fleischer ring, 385
 of keratitis disciformis, 519
 of keratoconus, 355
 of lattice-like dystrophy, 339
 of lens, 70
 of lipin keratitis, 351
 of nodular dystrophy, 332
 of phlycten, 482
 of Tyndall phenomenon in anterior chamber, 564
- Dimmer's reticular dystrophy, 335
 keratitis nummularis, 461
- Dimples, Fuchs', of cornea, 306
- Dionine, 134
 vital staining of lymphatics, 148
- Direct focal illumination, 69, 72
 of acne rosacea keratitis, 481
 of bullae, 109
 of conjunctiva, 135
 of conjunctivitis, 449
 of cornea, 73, 74
 projection of shadows in, 108
 of corneal nerve fibers, 362
 of corneal nerves, 442
 of corneal scars, 537
 of edema of epithelium, 395
 of endothelium, 415
 of eyelashes, 257
 of eyelid margins, 252
 of fibrinous precipitates, 441
 of folds in Bowman's membrane, 411
 in Descemet's membrane, 421
 of Fuchs' dimples, 307
 of Herpes simplex corneae, 462
 of iris, 73
 of Kayser-Fleischer ring, 386
 of keratic precipitates, 435, 440
 of lens, 73, 74
 projection of shadows in, 108
 of limbal region, 145
 localization by focusing the microscope, 117
 of multiple epithelial erosions, 457
 of neuroparalytic keratitis, 489
 parallelepiped in, 74
 of pinguecula, 161
 of sclera, 73
 of siderosis of cornea, 391
 of Stähli-Hudson line, 385
 of synchysis scintillans, 594
 of Tyndall phenomenon in anterior chamber, 563
 of vascularization in interstitial keratitis, 502
 of vessels, corneal, 409
 of vitreous, 78
- Diseases, hemopoietic, 248, 251
- Distortion, primary, 115
 real, 115
- Droplets, of cornea, 146, 285
 direct focal illumination of, 119

Droplets— (*Continued*)

- of epithelial dystrophy, 318
- retro-illumination of, 80, 88, 119, 285

Drüsen, in white rings of cornea, 342

Dysostosis multiplex, 316

Dystrophia adiposa corneae, 316

Dystrophia corneae reticulata, 335

Dystrophy, alphabet, 337

- band-shaped opacity of cornea, 343

- circular peripheral opacity, 340

- classification of, 316

- constitutional, 309, 311

- of cornea, 302, 307, 316

- familial, 316, 324

- lattice-like, 311, 316, 324, 335

- macular, 316, 324, 332

- nodular, 316, 324, 325

- farinata, 342

- cornea guttata, 311, 319, 320

- marginal degeneration in, 363

- opacities in, 307

- of corneal stroma, 316

- bandform keratitis, 317, 343

- circular peripheral opacity, 316, 340

- cornea farinata, 316, 342

- dystrophia adiposa corneae, 316

- dysostosis multiplex, 316

- embryotoxon, 316

- hereditary crystalline degeneration, 316, 352

- Hurler's syndrome, 316

- keratitis nummularis, 316

- keratoconus, 316, 353

- lipin degeneration, 316, 346

- marginal dystrophy, 316, 363

- shagreen, 316, 342

- white limbus girdle, 317, 366

- rings of Coats, 316, 340

- whorl opacity, 316

- degeneration, calcareous, 368

- hyaline, 368

- ectatic marginal, 363

- edema, corneal, 309

- of endothelium, 310, 316, 320

- localization of, by zones of specular reflection, 121

- optic section of, 322

- epithelial, 316, 317

- diffuse, 317

- opacities, 318

- optic section in, 318

- symmetric partial, 317

Dystrophy— (*Continued*)

- erosions, multiple recurring, 317

- familial corneal, 324

- "fat," of cornea, 352

- Fleischer's ring, 325

- Fuchs', 309, 317

- grill-like keratitis, 335

- Groenouw nodular, 324

- hereditary, of cornea, 309, 311

- keratoconus, 311, 353

- lattice-like type, 335

- diffuse illumination of, 339

- optic section of, 339

- parallelepiped in, 339

- retro-illumination of, 339

- sclerotic scatter in, 339

- tears in Bowman's membrane in, 412

- vital staining of, 338

- lipin keratitis, 311, 346

- macular type, 332

- optic section in, 335

- parallelepiped in, 335

- retro-illumination of, 335

- sclerotic scatter in, 335

- nodular (Groenouw), 324

- diffuse illumination of, 332

- optic section in, 332

- parallelepiped in, 332

- retro-illumination of, 325, 332

- sclerotic scatter in, 332

- opacity, bandformed, 343

- of parenchyma, 311, 352

- reticular (Dimmer), 335

- shagreen in, 342

- superficial, honeycomb effect, 411

- vortex, of cornea, 371

- white limbus girdle, 366

- Ectasia, acute, in ruptures of Descemet's membrane, 361

- of cornea, 363, 532

- thinning of, in, 445

- Ectatic marginal dystrophy, of cornea, 363

- Ectropion, in blepharitis, 261

- in keratitis e lagophthalmos, 476

- in xerosis, 488

- Edema, of conjunctiva, 176

- of cornea, 285, 393

- in dystrophies, 309

- Edema—(*Continued*)
 of cornea—(*Continued*)
 and folds in Descemet's membrane, 414
 of corneal epithelium, retro-illumination and direct focal illumination of, 117
 of endothelium, 415
 of epithelium, 394
 increase in corneal thickness in, 444
 in neuroparalytic keratitis, 489
 parallelepiped in, 395
 parenchymal, 414
 retro-illumination of, 88, 91, 395, 399
 specular reflection of, 395
 Ehrlich-Turk line, 566, 573
 Elliot trephining operation, 647
 Elschnig screw for biomicroscope, 24
 Embryotoxon, 297, 301, 316
 in megalocornea, 291
 in microphthalmos, 290
 Endothelium, direct focal illumination of, 415
 retro-illumination of, 415
 Entropion, 210
 Epinephrine, effect of, on conjunctival circulation, 134
 Episclera, nodule in, in herpes zoster keratitis, 466
 Episcleritis, 270
 biomicroscopy of, 270
 fugax, 270
 optic section in, 270
 periodica fugax, 229
 Epitarsus, 155
 Epithelial bullae of cornea, 399
 Epithelial changes in parallelepiped, 449
 Epitheliolysis, 286
 Epithelioma, basal-cell, of eyelid margin, 265
 of conjunctiva, 236
 of cornea, 236
 Epithelium, edema of, 395
 direct focal illumination of, 395
 erosions in, 400
 filaments in, 400
 fissures, 400
 intra-epithelial foci in, 400
 punctate dots in, 400
 subepithelial lines, 400
 Erythema multiforme (exudativa), 223
 Erythema nodosum, 223
 Examination, of conjunctiva, 134
 of cornea, 392
 of eyeball, 64
 illumination of room for, 64
 procedure, 122
 of transparent ocular media, 9
 of zones of specular reflection, 91
 of corneal surface, 97
 Exposure keratitis, 471
 Eye, transparent ocular media of, 9
 Eyeball, oscillations of, 31
 survey of, preliminary, 64
 Eyelashes, 257
 direct focal illumination of, 257
 Eyelid margin, 252
 biomicroscopy of, 252
 blepharitis, chronic, 260
 chalazion, 261
 cilia, 257
 Eyelids, direct focal illumination of, 252
 erythema nodosum of, 223
 eyelashes, 257
 fluorescence of, 18
 hordeolum externum, 261
 hyperemia of, 260
 papillomatous excrescences on, 261
 parasitic infection of, 265
 pathology of, 260
 zones of, 253
 Facets, 212, 306
 Facies, Hutchinson's, 492
 Fehr's macular corneal dystrophy, 332
 Fibrinous precipitates, direct focal illumination of, 441
 retro-illumination of, 441
 Fibromas, of conjunctiva, 251
 Filaria loa, 236
 Film line, 137
 optic section of, in corneal ulcer, 525
 Filtration angle, 371
 Fincham biomicroscope, 53
 Fixation, 31
 Fleischer's ring, 325, 362
 Fluorescein, 289
 in acute conjunctivitis, 185
 administration of, 18
 in arcus senilis, vital staining of, 303
 in conjunctiva, vital staining of, 137

Fluorescein—(*Continued*)

- in corneal erosions, 543
- in corneal wounds, 548
- in cysts, conjunctival, 235
- in epithelial changes, 400
- in filamentary keratitis, 491
- in herpes simplex corneae, 463
- in herpes zoster keratitis, 466
- in keratitis e lagophthalmos, 471
- in keratoconus, 363
- in meibomian glands, vital staining of, 257
- in multiple epithelial erosions, 457
- in neuroparalytic keratitis, 489
- in phlycten, 482
- in pinguecula, vital staining of, 165
- in precorneal film, 281
 - line, epithelial and subepithelial changes in, 449
- in sclerocorneal tuberculosis, 511
- in superficial linear keratitis, 461
- in thinning of cornea, vital staining of, 445

Fluorescent light, 18

Focal beam, in examination of, anterior chamber contents, 564

- of retina, 5
- rectangular, 6

Focal illumination, 5, 9. (*See also* Direct focal illumination)

- of corneal stroma, 2
- direct, 69
- hand slit lamp in, 58
- of superficial vortical pigmentation of cornea, 372

Folds, of Bowman's membrane, 411

- in direct focal illumination, 411

- of Descemet's membrane, 417
 - diffuse illumination of, 421
 - direct focal illumination of, 421
 - in herpes simplex corneae, 463
 - retro-illumination of, 421

Follicles, of conjunctiva, 139, 183, 190

- biomicroscopy of, 183
- differentiation of, 183, 194

Follicular conjunctivitis, 194

Foreign bodies, in anterior chamber, 586

- in angle of anterior chamber, 615
- caterpillar hairs, 587
- cilia, 586
- metallic, 586
- in conjunctiva, 230

Foreign bodies—(*Continued*)

- in cornea, 548, 553
 - folds in Bowman's membrane, 411
 - metallic, 554

Fornix, biomicroscopy of, 139

Fuchs' dimples, 306

- diffuse illumination of, 307
- direct focal illumination of, 307
- epithelial dystrophy, 317
- senile marginal atrophy, 363
- superficial punctate keratitis, 450

Fundus, axis of illumination in examining, 66

of observation, 66

Fungus infections, of canaliculi, 269

- of cornea, 531

Gargoylism, 316

lipin keratitis in, 351

Gas, mustard, 556

Gasserian ganglion, lesion of, in herpes zoster keratitis, 466, 467

- in neuroparalytic keratitis, 488

Gaucher's disease, 165

Gerontoxon, 302

Glass membranes, 409, 578

- folding of, in corneal wounds, 550
- reduplication of, 580
- specular reflection of, 105

Glass-like membranes, in interstitial keratitis, 500

Glaucoma, 630, 635

- acute, 632
 - alterations following iridectomy, 651
 - congestive phases, 652
 - cyclodialysis, 655
 - hyperopia in, 611
 - shallow chamber angle in, 632
- anterior chamber, shallow, in, 632
- capsulare, 638
- cataract surgery in, 659
- chronic congestive, 632
 - alterations following operations for, 651
 - cyclodialysis, 658
- chronic simple, 635
- congenital, 291, 627
- congestive, 632
- cyclodialysis for, 654
- diabetic rubeosis iridis in, 641
- dislocated lens in, 641
- filtration angle in, 371

Glaucoma—(*Continued*)

- gonioscopy, value of, in, 617
 - goniotomy for, 670
 - hemorrhages in, 641
 - infantile, 295
 - iridectomy for, 651
 - iris inclusion operations for, 666
 - Lagrange sclerectomy for, 666
 - lenticular intumescence in, 640
 - mydriatic in, 611
 - narrow-angle, 632
 - alterations following operations for, 651
 - operations for, 650
 - goniotrabeculotomy for, 599
 - simplex, 634
 - depth of angle of anterior chamber in, 610
 - size of eye in, 611
 - subluxated lens in, 640
 - synechiae, 599, 630
 - treatment of, 633, 653
 - trepining operation for, 660
- Goldmann (Haag-Striet) biomicroscope, 56
- Goniometer, 121
- Goniometry, 609
 - measurement of chamber angle acuity by, 608
- Gonioscope, Troncoso, 606
- Gonioscopy, apparatus for, 599
 - binocular corneal microscope in, 606
 - clinical value of, 614
 - contact lenses for, 600
 - in cyclodialysis, 654
 - examination by, 619
 - of chamber angle, 596, 598
 - in megalocornea, 291
 - of filtration angle, 371
 - history of, 598
 - in iridectomy, 651
 - in iris inclusion operations, 666
 - Koeppé "A" lens in, 599, 601
 - Koeppé "C" lens in, 599, 601
 - in Lagrange sclerectomy, 666
 - lenses for, 599
 - in photography of anterior chamber angle, 609
 - technique of, 599
 - in trephine openings, 660
- Goniotomy, 670
- Goniotrabeculotomy, 599

- Granuloma, of conjunctiva, 251
- Greenough microscope, 29, 32
- Groenouw's familial corneal dystrophy, 324, 332
- Gullstrand, Allvar, 1
 - illuminating system of, 20
 - Vogt method, difference between, 25
 - slit lamp of, 7, 14
- Gumma of conjunctiva, 219
- Haab's and Dimmer's reticular degeneration of cornea, 335
- Haab's striae, 296
- Haag-Striet biomicroscope, 56
- Hairs, caterpillar, in anterior chamber, 587
 - in conjunctivitis, 227
 - in cornea, 228
- Hand slit lamp, 58
 - Bausch and Lomb, 59
 - Bettman and McNair, 62
 - Bishop Harman, 62
 - Featherweight, 58
 - Krimsky, 58
 - May ophthalmoscope, 58
 - Wappler U-shaped hair-line filament, 58
- Hartinger, images, primary, 25
 - formula, 120
- Hartnack microscope, 2
- Hassall-Henle bodies of cornea, 279, 305
- Hemangioma of conjunctiva, 248
- Hemipoietic system, 248
- Hemorrhage, of conjunctiva, 137, 168
 - due to injury, 229
 - red free light, in examination of, 137
- Henker's arm, 33
- Henker's biomicroscope, 8, 14
- Herbert's marginal pits, 212
- Hereditary crystalline degeneration, 316, 352
- Herpes simplex corneae, 461
 - biomicroscopy of, 462
 - direct focal illumination of, 462
 - optic section of, 463
- Herpes zoster, 466
 - keratitis, scleritis in, optic section of, 466
 - ophthalmicus in conjunctiva, 200
- Histamine, 134

- History, of biomicroscopy, 1
 of corneal microscope, 4
 of gonioscopy, 599
 of oblique illumination, 1, 2
- Hordeolum externum, 261
- Hudson's line, 381
- Hurler's syndrome, 316, 347, 351
- Hutchinson's facies, 492
- Hyaline membrane, in anterior chamber, 569, 572, 578
 in birth injuries, 578
 in interstitial keratitis, 500
 specular reflection of, 105
 striae, 416, 422
 optic section of, 416
- Hydroa vacciniiform, 227
 conjunctivitis in, 227
 corneal complications in, 227
- Hydrogen peroxide, in vital staining of lymphatics, 148
- Hydrophthalmos, 295
 congenital alteration in chamber angle, 627
- Hyperemia, 260
 in acne rosacea, 228
 in allergic conjunctivitis, 190
 of conjunctiva, 176
 due to injuries of, 229
 of eyelid border, 260
- Hypersensitivity, tuberculo-allergic, 190
- Hypertension, aneurismal distentions of peripheral vessels in, 133
- Hypphemia, absorption of, 577
 of anterior chamber, deposits on cornea in, 439
- Hypopyon, 524
 in herpes simplex corneae, 465
- Illuminating beam, angle between, and axis of microscope, 66
- Illuminating lens, 22
 Arruga screw, 24
 Elschnig screw, 24
 focal length of, 23
 Vogt diaphragm aperture, 23
- Illuminating system, in biomicroscope, 14
 condensing system in, 20
 diaphragm in, 20
 Koeppel's tube in, 21
 plano-convex cylinders in, 21
 in Poser biomicroscope, 47
- Illumination, 69
 afocal, of corneal nerves, 277
 arc lamp, 15, 24, 25
 lenses with, 23
 axis of, 66
 of biomicroscope, 69
 carbon arc lamp, 16
 co-axial, in gonioscopy, 606
 cold light lamp, 137
 condensed focal, 1
 darkfield, 83
 diffuse, 29, 69. (*See also* Diffuse illumination)
 direct focal, 72. (*See also* Direct focal illumination)
 of examining room, 64
 focal, 6, 9
 beam, 5
 in examining retina, 5
 condensed, 1
 Gullstrand method of, 20
 Vogt method, differences between, 25
 indirect, 105. (*See also* Indirect illumination)
 internal trans-scleral, 79
 in intrinsic specularity, 103
 Koeppel system of, 38, 40
 Kohler-Vogt method of, 8, 20
 magnifications, high, of, 31
 fixation in, 31
 mercury vapor lamp in, 16
 method, of Gullstrand, 25
 of Kohler-Vogt, 20
 specular reflection with, 97
 of Vogt, 25
 in narrow beam, 78. (*See also* Narrow beam)
 with nernst lamp, 25
 in projection of shadows, 108
 proximal, 105. (*See also* Indirect illumination)
 with quartz ultraviolet lamp, 18
 red free, 137
 in reflection, structureless, 105
 retro-illumination, 69, 79. (*See also* Retro-illumination)
 direct, 83
 indirect, 83
 localization, 91
 reversed, 321

- Illumination—** (*Continued*)
 of room, 64
 sclerotic scatter, 69, 70, 88. (*See also* Sclerotic scatter)
 source of, 14
 in Universal slit lamp, Koeppe model, 38
 specular reflection, 69, 91. (*See also* Specular reflection)
 unreversed, 90
 in bullae, 399
 with yellow filter, 137
- Images, primary,** 24
 luminous intensity of, 24
 variations in, 24
- Incisions, surgical, of cornea,** 551
- Inclusion blennorrhea,** 200
- Indirect illumination,** 69, 105
 of conjunctiva, 136
 of corneal nerves, 277
 of edema of epithelium, 395
 examination with, 105
 of keratic precipitates, 440
 of limbal region, 145
 in macular dystrophy, 335
 of marginal ulcer of cornea, 529
- Inflammation, of conjunctiva,** 176
 of cornea, 447
 deep zone of, 492
 superficial zone of, 448
- Injection, of conjunctiva,** 176
 of cornea, 402
- Injuries, of anterior chamber,** 572
 of chamber angle, 642
 of conjunctiva, 229
 biomicroscopy of, 229
 burns, 230
 cysts, 235
 epithelialization, 229
 foreign bodies, 230
 hemorrhage, 229
 incised wound, 230
 of cornea, 424, 541, 547
 tears of Bowman's membrane, 412, 422
 traumatic, 541
 wounds, 547
- Insect stings, of cornea,** 557
- Internal specularity, of cornea,** 276
 of corneal stroma, 103
 direct focal illumination of, 103
 of lens, 103
- Interstitial keratitis,** 492. (*See also* Keratitis)
- Intrinsic optic properties of ocular media,** 11
 specularity, 103
 direct focal illumination in, 103
- Iridectomy,** 651
- Iridencleisis,** 667
- Iris, diabetic rubeosis,** 641
- Iridocyclitis, epithelial keratitis associated with,** 456
 keratic precipitates in, 426, 433
- Iridotaxis,** 667
- Iris, bombé, anterior chamber in,** 595
 coloboma, 570
 congenital, 620
 direct focal illumination of, 73
 indirect illumination of, 105
 neoplasms in, 593
- Iris inclusion operations,** 653
 synechiae, anterior, in, 595
- Iritis, acute, precipitates in,** 441
 fetal, 569, 570
 folds in Descemet's membrane in, 417
 gonorrheal, keratic precipitates in, 434
 herpetic, 465
- Iron deposits, on conjunctiva,** 175
 on cornea, 391
- Kayser-Fleischer ring,** 385
 diffuse illumination of, 386
 direct focal illumination of, 386
 retro-illumination of, 386
- Keloids, cutaneous,** 539
- Keratic precipitates, biomicroscopy of,** 426, 440
 differential characteristics of, 441
 direct focal illumination of, 435, 440
 distribution of, 429
 in dystrophy, 309
 in herpes simplex corneae, 463
 in herpes zoster keratitis, 466
 in keratitis, interstitial, 494
 localization of, 119, 426
 in melanin pigment, 370
 narrow beam in, 427
 physiologic deposits, 434
 pigment deposits, 435
 retro-illumination of, 90, 435, 440
 indirect, 90, 440
 sclerotic scatter of, 440
 in uveitis, 441

Keratic precipitates— (*Continued*)

vital staining of, 289

Keratinization, of conjunctiva, 189

Keratitis, acne rosacea, 476

nodular type, 477

anaphylactica, 492

annularis, 500

bandform, 317, 343, 534

honeycomb effect, 411

bullous, 399

central, 352

folds in Bowman's membrane in,
411centralis annularis (Vossius), 414, 500,
507

diffuse epithelial, 456

diffuse illumination of, 456

diffuse retro-illumination of, 351

disciformis, 519

folds in Descemet's membrane in,
417

increase in corneal thickness in, 444

superficial punctate keratitis and,
451

vascularization in, 407

eczematosa, 385

e lagophthalmos, 471

ephemeral superficial punctate, 458

retro-illumination of, 458

epithelial, diffuse, 456

with iridocyclitis, 456

erosions, multiple epithelial, 457

exposure, 471

fädchen, 470, 490

fascicular, 482

retro-illumination of, 482

filamentary, 490

differentiation from keratoconjunc-
tivitis sicca, 470

filamentosa, 490

filiformis, 490

grill-like, 335

herpes febrilis, 461

herpes simplex, 461

herpes zoster, 466

iritis in, 467

ulceration in, 466

vital staining of, 466

herpetic, 400, 449, 461

folds in Descemet's membrane in,
417

keratic precipitates in, 433

Keratitis— (*Continued*)

hyperplastic, 515

interstitial, 214, 492

atypical, types of, 506

avascular, 503

blood staining of cornea in, 377

corneal thickness in, 444

diffuse, syphilitic, 503

palisades in, 146

folds in Descemet's membrane in,
417

hyaline striae in, 416

keratic precipitates in, 434

opacification in Descemet's zone in,
414

optic section of, 494

reparation in, 501

salmon patch in, 408

scars in, 539

sequelae of, 506

syphilitic, 503

salmon patch in, 501, 510

thinning of cornea in, 445

optic section of, 508

tuberculous, 510

vascularization in, 401, 403

vessels, retro-illumination of, in, 502

zones of specular reflection, localiza-
tion by, in, 121

lattice-form, 335

Leishmania, 518

leprous, 515

hyperplastic, 515

linear, superficial, 459

folds in Bowman's membrane in,
411

lipin, 311, 346

marble-like, 458

marginal, vascularization in, 408

marmorata, 458

metabolic disorders in, 468

metaherpetica, 467

neuromparalytic, 488

in herpes zoster keratitis, 467

retro-illumination of, 489

neurotrophic disorders in, 468

nummularis of Dimmer, 316, 461

parenchymatous, 467, 492. (*See also*
Keratitis, interstitial)

annularis of Vossius, 506

infiltrative, 515

peripheral furrow, 363

Keratitis— (*Continued*)

- petrificans, 343
- phlyctenular, 468, 481
 - pannus in, 483
 - ulceration in, 482
 - vascularization of, 401, 403
 - vessels in, retro-illumination of, 408
- physical disorders in, 468
- profunda, 352, 518
 - vascularization in, 407
- punctata profunda, 506
- punctate, superficial, 450
 - sclerotic scatter of, 451
 - superficial ephemeral, 458
- rosacea, 468, 476
 - thinning of cornea in, 446
 - vascularization in, 403
 - vessels in, 408
- sclerosing, 513
- striate, folds in Descemet's membrane in, 421
- superficial inflammatory, 448
 - linear, 459
 - punctate, 450
 - retro-illumination of, 456
- suppurative, 520
- tuberculous, infiltration in, 414
- vaccinial, 468
- varicella, 468
- vascularized, 401
- vesicular, diffuse, 488
- Keratoconjunctivitis eczematosa, 481
 - epidemic, 195
 - erythema multiforme, 223
 - Parinaud's syndrome in, 221
 - phlyctenular, 190, 481
 - sicca, 470
 - differentiation of, from filamentary keratitis, 491
 - optic section of, 471
 - retro-illumination of, 471
 - vital staining of, 471
- Keratoconus, 311, 316, 353
 - corneal stroma in, striae in, 414
 - Descemet's membrane in, ruptures of, 360
 - tears in, 422
 - diffuse illumination of, 355
 - endothelial cup in, reflex of, 355
 - Fleischer's ring, 362
 - nerve fibers in, 362

Keratoconus— (*Continued*)

- opacities in, 360
- posticus, 297, 363
- scars in, 360
- striae in, 355
- thinning of cornea in, 355, 445
- Keratomalacia, 483
- Keratomegalia, 291
- Keratomycosis, 531
- Kleefeld silvered mirror, 66
- Koeppel, "A" lens, 601
 - "C" lens, 601
- contact glass, 596
- method of examining anterior chamber angle, 597
- microscope, 28
- silvered mirror, 50
 - in examination of anterior chamber angle, 597
- system of illumination, 38, 40
- tube, 21
 - plano-convex cylinders, 21
- Kohler-Vogt method of illumination, 8, 20
- K.P., 426. (*See also* Keratic precipitates)
- Krause's corpuscles, 149
- Krimsky hand slit lamp, 58
- Krukenberg's spindle, 369, 371, 372
 - keratic precipitates in, 433
- Lacerations, of cornea, 550
- Lacrimal caruncle, 147
 - biomicroscopy of, 147
- Lacrimal puncta, 265
 - canaliculi, 269
 - inflammation of, 269
 - vital staining of, 269
- Lagrange sclerectomy, 652
- Lamp, arc, 15
 - lenses for, 23
- carbon arc, 16
- mercury vapor, 16
- ernst, 7, 14
 - intensity of, 24, 25
- nitra, 14
 - intensity of, 24, 25
- nitrogen-filled, 52
- pointolite, 24, 25
 - intensity of, 24, 25
- quartz ultraviolet, 18
- slit, and corneal microscope, 2

- Layers, of transparent ocular media, examination of, 9
- Lemoine and Valois biomicroscope, 53
- Lens, blebs in, optic section of, 109
- block, 76
- capsule, specular reflection of, 102
- hyaline membranes of, 105
- cataract, focal illumination of, 74
- retro-illumination of, 79
- contact, 50
- diffuse illumination of, 70
- direct focal illumination of, 73, 74
- internal specular reflection with, 103
- focusing of beam on, 77
- fluorescence of, 18
- growth of, shallow chamber angle in, 610
- hand slit lamp, in examination of, 58
- illuminating, 22
- measurement of, with Ulbrich micrometer, 119
- narrow beam of, 78
- opacity of, in direct focal illumination, 117
- retro-illumination of, 81
- optic section of blebs and vacuoles in, 109
- pigment on anterior capsule of, projection of shadows in, 108
- plastic, for biomicroscopy, 600
- posterior pole of, axis of beam in examining, 2
- axis of observation in examining, 2
- Purkinje, 1
- relucency of, 80
- retro-illumination of, 79, 83
- thickness of, micrometry of, 120
- vacuoles in, optic section of, 109
- zones of discontinuity, 23, 103
- direct focal illumination of, 76
- zones of discontinuity of, in age, 103
- specular reflection in, examination for, 91
- Leproma, of conjunctiva, 219
- Leprosy, of conjunctiva, 219
- conjunctivitis in, 219
- of cornea, 515
- keratitis in, 515
- lepromas, 219
- Leukoma, in atheromatous ulcer, 530
- of cornea, 107, 532
- adherent, 532
- narrowing of anterior chamber in, 595
- congenital, 299
- indirect illumination of, 107
- Lichen scrofulosus, 214
- Light, focusing of, 65
- fluorescent, 18
- reflexes, 91
- intensities of sources of, 25
- intensity, loss of, 74
- sources, of Poser biomicroscope, relative difference of brilliance of, 52
- Limbal arcades, 402
- Limbal palisades, 146
- Limbal region, 145
- direct focal illumination of, 145
- optic section of, 145
- retro-illumination of, 145
- Limbal spur, 285
- biomicroscopy of, 127, 145
- optic section of, 393
- Limbal vessels, 127
- Limbus, biomicroscopy of, 145
- vascular arcades in, 127
- Line, Arlt's, 207
- Ehrlich-Turk, 433, 566, 573
- Fleischer's, 362
- Hudson's, 362, 381
- Stähli's, 362, 381
- Turk's, 369, 371
- Linea corneae senilis, 362, 381
- Lipin degeneration, primary, 316
- Lipin interstitial keratitis, 352
- Lipin keratitis, 346
- diffuse illumination of, 351
- optic section of, 351
- Localization, 111
- of anterior chamber, 120
- direct focal illumination in, 117
- distortion, primary, in, 115
- distortion, real, in, 115
- of droplets of edema of corneal epithelium, 119
- by focusing of microscope, 117
- of keratic precipitates, 119
- methods of, 111
- micrometry in, 120
- of neovascularization, 408

- Localization—(*Continued*)
 by optic section, 111
 in epithelial and subepithelial changes, 448
 by parallax displacement, 122
 by projected shadows, 121
 in retro-illumination, 91
 in specular reflection, 91
 Loupe, binocular, 5
 and lens, 5
 Lucanus curved rail, 4
 Luminous beam, optical characteristics
 of, 24
 intensity of primary images in, 24
 Lupus, of conjunctiva, 509
 of cornea, 509
 Lupus erythematosus, 214
 of conjunctiva, 214
 Lymphangiectasis, 250
 Lymphangioma, 250
 optic section of, 250
 Lymphatics, 148
 biomicroscopy of, 148
 vital staining of, 148
 Lymphogranuloma venereum, 221
 Lymphoma, of conjunctiva, 250

 Macula, in cornea, 532
 Maculae, in interstitial keratitis, 494
 in sclerotic scatter, 494
 Madarosis, 210
 Magnification, 31
 low, 67
 with paired objectives, 41
 Malignant tumors, of conjunctiva, 243
 Marginal dystrophy, 316, 363
 of cornea, 363
 oblique illumination of, 365
 optic section of, 366
 parallelepiped in, 365
 ectatic, 363
 Mayou biomicroscope, 56
 Megalocornea, 291
 congenital, 627
 deepening of anterior chamber in, 568
 Megalophthalmos, anterior, 291, 568
 Meibomian glands, 257
 Melanin, 168
 in herpes zoster ophthalmicus, 201
 pigment, in cornea, 369, 370

 Melanoma, malignant, of conjunctiva, 243
 Melanosarcoma, of uveal tract, 248, 593
 Melanosis, of conjunctiva, 247
 Melanosis oculi, 155, 247
 optic section of, 155
 Membranes, glass, 578
 hyaline, 578
 pseudo-, 589
 pupillary, 569
 structureless, specular reflection from, 105
 Mercurochrome, 286
 Mercury vapor lamp, 16
 Metaplasia, partial, of conjunctiva, 154
 Methylene blue, 286, 288, 289
 in vital staining, of conjunctiva, 149
 of conjunctival epitheliomas, 237
 in herpes simplex corneae, 463
 Microcornea, 290
 anterior chamber in, 569
 optic section in, 290
 Micrometer, corneal thickness with, 444
 Ulbrich, 119
 Micrometry, 120
 Microphthalmos, 290
 shallowing of anterior chamber in, 569
 Microscope, 28
 Abbé, 28, 32
 Aubert, 29
 axis of, angle between illuminating beam and, 66
 binocular, 28, 52
 corneal, Aubert, 2
 Aubert-Czapski, 2, 4
 in gonioscopy, 606
 Greenough type, 37
 monobjective, 28
 bitumi (Seidentopf), 32
 corneal, Aubert, 2
 Aubert-Czapski, 2, 4
 history of, 4
 binocular, 28
 Czapski, 2, 8, 14, 28, 32
 Czapski-Schatz, 29
 Czapski-Zeiss, 32
 disadvantages of, 34
 focus of, in specular reflection, 97
 focusing of, in localization, 117
 Greenough, 29, 32
 Hartnack, 2

Microscope—(*Continued*)

- Henker's arm, 33
- history of, 28
- Koeppe, 28
- monobjective, binocular, 28
- monocular, 28
- ortho-bitumi, 28, 32
- Schick, 2
- Seidentopf modification of, 28
- ultra-, 10
- Zeiss, 34
- Mirror, Kleefeld silvered, 66
 - Koeppe silvered, 50
- Mooren's ulcer, 529
 - optic section of, 530
- Multiple epithelial erosions, direct focal illumination of, 457
- Mustard gas, corneal damage by, 556
- Mutton-fat precipitates, 442
- Myopia, anterior, 353
- Narrow beam, 78
 - in acne rosacea keratitis, 477
 - in anterior chamber, 595
 - in conjunctival cysts, 235
 - in contusions, 585
 - in corneal stroma, 78
 - scars, 532
 - importance of, 79
 - in keratic precipitates, 427
 - in lens, 78
 - in neovascularization of conjunctiva, 178
 - optic section with, 78
 - in papillae of conjunctiva, 178
 - in scleral plaques, 272
 - in sclerocorneal tuberculosis, 511
- Nebula, of cornea, 532
 - in Bowman's membrane, 411
 - in phlyctenular keratitis, 482
 - retro-illumination of, 88
- Neoformation, 401
 - of conjunctiva, using narrow beam, 178
 - localization of, 408
 - in nevi of conjunctiva, 243
 - optic section of, 408
 - retro-illumination of, 408
 - of vessels, 402
- Nernst lamp, 6, 14
 - intensity of, 24, 25
- Nerves, corneal, 277
 - increased visibility of, 442
 - conjunctival, vital staining of, 149
- Neuro-ectodermal tumors, 240
- Neuroparalytic keratitis, 488
 - direct focal illumination of, 489
 - optic section of, 489
 - retro-illumination of, 489
- Nevi, of eyelid margin, 265
- Nevus, of conjunctiva, 242
 - malignant, 243
 - optic section of, 247
 - of cornea, 242
 - flammeus, 249, 260
- Nile blue sulphate, 288
- Nitra lamp, 14
 - brilliance of, 52
 - intensity of, 24, 25
- Nitrogen-filled lamp, 52
- Nodular dystrophy, of cornea, Groenouw's, 324
 - diffuse illumination of, 332
- Nodules, in acne rosacea, 228
 - in sclerocorneal tuberculosis, 511
- Objectives, paired, 44
 - magnifications of, 44
- Oblique illumination, 9
 - Aubert low voltage electric lamp in, 2
 - cataracts in, 2
 - Helmholtz on, 1
 - history of, 1, 2
 - Liebrich technique of, 2
 - marginal degeneration of cornea in, 365
 - with monocular microscope, 28
 - opacities in, 2
- Observation, axis of, 66
- Occlusio pupillae, 595
- Ochronosis, 169
- Oculoglandular tularemia, 220
- Opacities, in aqueous, 589
 - in Bowman's membrane, 410
 - cobweb, in aqueous, 589
 - of cornea, 410, 531
 - bandformed, 343
 - congenital, 298
 - irregular superficial, in keratoconus, 360
 - leukomatous, 510
 - ribbon-shaped, 343

- Opacities—(*Continued*)
 of cornea—(*Continued*)
 striate, grill-like, 537
 whorl, 371
 zonular, 343
 in lens, retro-illumination of, 80
 reticular, in aqueous, 589
- Operations, tears in Descemet's membrane in, 424
- Ophthalmia nodosa, 227
- Ophthalmomicroscope, de Wecker, 2
 Gayet, 2
- Ophthalmoscope, Wolff, 5
- Optic border zone, 565
- Optic properties of structures, obstructive, 88
 refractile, 88
 respersive, 88
- Optic reduplications, 108
- Optic section, 111, 446
 of acne rosacea keratitis, 477
 of anterior chamber, 563
 of anterior corneal surface, 278
 of arcus senilis, 303
 of argyrosis of cornea, 391
 of bandformed opacity, 346
 of blood staining of cornea, 377
 of Bowman's membrane, 410
 of bulbar conjunctiva, 140
 of bullae, 399
 of buphthalmos, 296
 of chalazion, 261
 of conjunctiva, 135
 epithelial layer of, 137
 of cornea, 279
 in localization, 112
 with narrow beam, 78
 observational distortion in, 115
 primary distortion in, 115
 refractile distortion in, 115
 of corneal abrasions, 543
 nerves, 277
 opacities in keratoconus, 360
 scars, 532
 thickness, 444
 in corneal ulcer, 523
 wounds, 549
 of cysts, implantation, 580
 of deep vessels in cornea, 409
 of dermoid tumors, 155
 of Descemet's zone, 415
 of endothelial dystrophy, 322
- Optic section—(*Continued*)
 in episcleritis, 270
 of epithelial dystrophy of Fuchs, 318
 of film line in corneal ulcer, 525
 of follicular conjunctivitis, 195
 of foreign bodies in cornea, 553
 of Fuchs' dimples, 306
 of herpes simplex corneae, 463
 of herpes zoster keratitis, scleritis in, 466
 of hyaline striae and excrescences, 416
 incidence of slit beam in, 286
 of injury of cornea, 424
 of internal specularity of cornea, 285
 of interstitial keratitis, 494
 atypical, 506
 avascular, 503
 in thinning of cornea, 508
 of keratoconjunctivitis sicca, 471
 of keratoconus posticus, 297
 of lattice-like dystrophy, 339
 of limbal region, 145
 of lipin keratitis, 351
 of lymphangiomas, 250
 of macular dystrophy, 335
 of malignant nevus, 247
 of marginal degeneration of cornea, 366
 of melanosis oculi, 155
 of microcornea, 290
 of Mooren's ulcer, 530
 of multiple epithelial erosions, 457
 narrow beam in, 78
 of neovascularization, 408
 of nodular dystrophy, 332
 of pannus trachomatosa, 211
 of phlyctenular conjunctivitis, 192
 of phlyctenular keratitis, 483
 of precorneal film line, vital staining of, 287
 in scleritis, 271
 brawny, 272
 in Stähli-Hudson line, 385
 of subacute and chronic conjunctivitis, 189
 of subepithelial layer of conjunctiva, 138
 of tarsal conjunctiva, 138
 of tear film line, 137
 of thinning of the cornea, 355
 in corneal scars, 445

Optic section—(*Continued*)

- of trachoma I, 202
- of trachoma II, 203
- of vacuoles, 109
- of vernal conjunctivitis, 193
- of white rings of cornea, 340

Optical block, 74

Optical characteristics of luminous beam, 24

Ortho-bitumi microscope, 32

Oscillations of eyeball, 31

Oscillatory illumination, 108

Osmic acid, 289

Palisades, biomicroscopy of, 146

Palpebral conjunctiva, 137

Pannus, 194, 403, 483

leprous, 515

in phlyctenular keratitis, 483

trachomatosa, 210

optic section of, 211

retro-illumination of, 211

sclerotic scatter of, 211

trachomatous, 210

corneal involvement in, 211

secondary degenerative changes in, 213

vessels in, retro-illumination of, 408

Panophthalmitis, 219

in keratitis e lagophthalmos, 476

Papillae, of conjunctiva, 178, 189, 194, 221

biomicroscopy of, 178

narrow beam in, 178

in vernal conjunctivitis, 192

vital staining of, 178

Papilloma, of conjunctiva, 231

Papillomatous excrescences, 261

Parallactic displacement, localization by, 122

Parallelepiped, 74

anterior corneal surface in, 278

in argyrosis of cornea, 391

in Bowman's membrane, 410

in bullae, 400

in cornea farinata, 342

in cornea guttata, 320

corneal, 74

nerves, 277

scars, 532

transparency, diminution of, 290

Parallelepiped—(*Continued*)

of depth of anterior chamber, 559

in direct focal illumination, 74

of edema of epithelium, 395

of epithelial and subepithelial changes, 449

in examination of anterior chamber, 564

incidence, of slit beam in, 286

of lattice-like dystrophy, 339

of macular dystrophy, 335

in marginal degeneration of cornea, 365

in nodular dystrophy, 332

of parenchymal cells of normal cornea, 288

of posterior surface of cornea, 279

in precorneal film layer, vital staining of, 287

in specular reflection, 94

of vessels, superficial, of cornea, 409

Parasites, in anterior chamber, 581

Parasitic infections, of eyelid margins, 265

Parinaud's syndrome, 220

biomicroscopy of, 221

conjunctivitis, follicular, in, 221

cysts in, 221

keratoconjunctivitis in, 221

lymphogranuloma venereum, 221

syphilis in, 221

Patient, management of, 65

preparation of, 65

Pemphigus, acute, 222

ankyloblepharon following, 223

biomicroscopy of, 222

bullae in, 222

cicatrization in, 222

of conjunctiva, 222

symblepharon in, 222

vulgaris, 222

Pericorneal injection, 402

Persistent pupillary membrane, 569

pigment deposits on cornea and, 435

Phenomena of fluorescence, 18

Tyndall, 10, 11

Phlycten in diffuse illumination, 482

Phlyctenular conjunctivitis, 190

optic section of, 192

Phlyctenular keratitis, 468, 482

optic section of, 483

Phosgene, corneal damage by, 556

- Phthiriasis palpebrarum, 265
- Physiologic deposits on cornea, 434
- Pigmentation, of conjunctiva, 154, 168
 in Addison's disease, 169
 bile, 168
 blood, 168
 endogenous, 168
 exogenous, 174
 melanin, 168
 in ochronosis, 169
 silver, 174
 in vitiligo, 173
 of cornea, 302, 369, 435
 hematogenous, 369, 373
 blood staining in, 373
 in Kayser-Fleischer ring, 385
 in Stähli-Hudson line, 381
 melanin, 369, 370
 in aged, 370
 congenital melanosis, 370
 in deposits, postoperative, 369
 traumatic, 369
 in dystrophies, 369
 in endothelium, 370
 epithelial, 370
 juxta-epithelial, 369
 keratic precipitates and, 369, 370
 in Krukenberg's spindle, 369, 371, 372
 in melanoma, 370
 in post-trachomatous pannus, 369
 caused by trachoma, 370
 in Turk's line, 369, 371
 in vortex dystrophy, 371
 in xerosis, 370
 metallic, 369, 387
 argyrosis, 387
 chalcosis, 391
 siderosis, 391
- Pilocarpine, 134
- Pinguecula, 161
 biomicroscopy of, 161
 direct focal illumination, 161
 optic section, 165
 vital staining, 165
 congenital, 155
 in Gaucher's disease, 165
- Plica semilunaris, 147
 congenital hypertrophy of, 155
- Pointolite lamp, intensity of, 24, 25
- Polychrome methylene blue, 286, 288
- Polyps, of conjunctiva, 251
- Porro system of erecting prisms, 28
 in Czapski binocular microscope, 29
- Poser biomicroscope, 41
 illuminating system of, 47
 light sources of, 52
- Posterior chamber, deepening of, 595
- Precipitates, keratic, 426
 mutton-fat, 433, 442
 retro-illumination of, 90
- Precorneal film line in keratoconjunctivitis sicca, 470
 vital staining of, with fluorescein, 449
 in optic section, 287
 in parallelepiped, 287
- Primary distortion, of optic section, 115
- Primary images, variations in luminous intensity of, 24, 25
- Prisms, porro system, 28
 in Czapski binocular microscope, 29
- Projection of shadows, 108
 localization by, 121
- Proximal illumination. *See* Illumination, indirect.
- Pseudopterygium, 235
 lime burns of cornea in, 556
- Pseudotrachoma, 215
- Pterygium, 305
 congenital, 155
- Ptosis, 210
- Punctum lacrimalis, 265
- Pupil, retro-illumination of, 79
- Pupillary membranes in anterior chamber, 569
 retro-illumination of, 79
- Purkinje lens, 1
- Quartz ultraviolet lamp, 18
- Red free light illumination, of conjunctival vessels, 137
- Reflection, irregular, 92, 93
 regular, 92
 specular, zones of, 69, 91
 from structureless membranes, 105
- Refractile distortion, of optic section, 115
- Refraction, errors of, in depth of anterior chamber angle, 611
- Rekoss diaphragm disk, 60

- Reticular degeneration, corneal, of Biber, Dimmer, and Haab, 335
- Retina, occlusion of central vein of, 641
 examination of, 50
 illumination of, focal beam, 5
- Retro-illumination, 69, 79
 of argyrosis, 391
 of bandformed opacity, 346
 of bedewing, 285
 of blebs, 446
 of cataract, 79
 of cells, stained, 288
 classification of, 83
 color of features in, 79
 of cornea, 79, 83, 408
 of cornea guttata, 311, 320
 corneal block in, 79
 corneal nerves in leprous keratitis, 518
 corneal vessels, 408
 in darkfield illumination, 83
 of diffuse epithelial keratitis, 456
 direct, 83, 90
 of cornea farinata, 342
 of cornea guttata, 320
 of keratic precipitates, 440
 sclerotic scatter in, 88
 of droplets, 80, 88, 285
 of dystrophy, symmetric partial epithelial, 317
 of edema of epithelium, 88, 285, 395
 of endothelial bedewing, 442
 of endothelium, 415
 of ephemeral keratitis, 458
 of epidemic keratoconjunctivitis, 198
 of fascicular keratitis, 482
 of fibrinous particles, 441
 of folds in Descemet's membrane, 421
 indirect, 83, 90
 of cornea farinata, 342
 of cornea guttata, 321
 of keratic precipitates, 90, 440
 sclerotic scatter in, 88
 of Kayser-Fleischer ring, 386
 of keratic precipitates, 435, 440
 of keratoconjunctivitis sicca, 471
 of lattice-like dystrophy, 339
 of lens, 79, 83
 opacity, 80
 of limbal region, 145
- Retro-illumination—(*Continued*)
 localization in, 91
 of macular dystrophy, 335
 of neovascularization, 408
 of neuroparalytic keratitis, 489
 of nodular dystrophy, 325, 332
 optic properties of structures in, 88
 of pannus trachomatosa, 211
 of pupillary membrane, 79
 of ruptures in Descemet's membrane, 361
 sclerotic scatter in, 88
 of Stähli-Hudson line, 381
 stenopeic pinhole in, 79
 of superficial punctate keratitis, 451
 of Tyndall phenomenon, in anterior chamber, 563
 of vacuoles, 90
 of vessels, in interstitial keratitis, 502
- Riboflavin, 401
 deficiency of, in acne rosacea keratitis, 476
- Ring, Fleischer, 362
 in keratoconus, 362
 Vossius, 414
- Rose Bengal, 286
 in vital staining, of conjunctiva, 137, 149
 in keratoconjunctivitis sicca, 471
- Rubeosis iridis, diabetic, 641
- Rupture, of Descemet's membrane, 542
 in keratoconus, 360
- Salmon patches, of interstitial keratitis, 408, 501, 510
 vascularization in, 407
- Scars, 110, 112
 corneal, 531
 in corneal ulcer, 525
 in keratoconus, 360
 in Mooren's ulcer, 530
 in optic section, 112
 in parallelepiped, 112
 due to surgical incisions, 551
 cystoid, 548
 herpetic, 533
 honeycomb effect, 411
- Schick microscope, 2
- Schlemm's canal, 617
- Schwalbe's ring, in goniometry, 609

- Scintillatio nivea, 594
- Sclera, direct focal illumination of, 73
indirect illumination of, 107
nodule in, in herpes zoster keratitis, 466
tuberculous lesion in, 511
- Scleral plaques, 272
narrow beam in, 272
- Scleritis, 271
annular, 271
biomicroscopy of, 271
brawny, optic section in, 271
in herpes zoster keratitis, 466
nodules in, 271
optic section in, 272
- Sclerocorneal tuberculosis, narrow beam in, 511
- Sclerokeratitis, 513
- Scleromalacia perforans, 272
- Scleroperikeratitis, 513
- Sclerotic scatter, 69, 70
acne rosacea keratitis by, 481
bandformed opacity by, 346
cornea by, 70
corneal nerves by, in leprous keratitis, 518
relucency by, 71
scars by, 532
faint by, 548
in direct retro-illumination, 88
in indirect retro-illumination, 88
keratic precipitates by, 440
keratitis disciformis by, 520
lattice-like dystrophy by, 339
maculae by, in interstitial keratitis, 494
macular dystrophy by, 334
marginal ulcer of cornea by, 529
nodular dystrophy by, 332
pannus trachomatosa by, 211
in retro-illumination, 88
superficial punctate keratitis by, 451
- Seclusio et oclusio pupillae, 595
- Seidentopf modification of Abbé microscope, 28, 32
- Senile arcus, 302
degenerations in, 302
- Shadows, artefact, 111
projection of, 108
- Shagreen, 316, 342
specular reflection in, 102
- Siderosis, of conjunctiva, 175
of cornea, 391
- Silver deposits, in conjunctiva, 174
in cornea, 387
- Silvered mirror, Kleefeld, 66
Koeppe, 50
- Slit lamp. (*See also* Biomicroscope)
hand, 58
simplified Universal, 38
Universal, 37
-Koeppe model, 37
- Sodium fluorescein, 286
- Specular reflection, 11, 91
catoptric images in, 92
of conjunctiva, 136
of cornea, 91, 94
of corneal surfaces, 97
of Descemet's membrane, 103
protuberances of, 416
tears in, 422
of endothelium, 416
of Hassall-Henle bodies, 305
of lens capsule, 102
from structureless membranes, 105
zones of, 69
corneal wounds in, 550
edema of epithelium in, 395
examination of, 91
localization in, 121
- Specularity, internal, of cornea, 276
- Spindle, Krukenberg, 369, 371, 372
keratic precipitates in, 433
- Spur, limbal, 127, 145, 285, 393
- Stähli-Hudson line, 362, 381, 537
direct focal illumination of, 385
etiology of, 385
optic section of, 385
retro-illumination of, 381
- Staphyloma pellucidum conicum, 353
- Stenopeic pinhole, 79
- Stereopsis, 68
- Stereoscopic effect in monobjective binocular microscope, 28
- Stings of cornea, 557
- Streptothrix, 269
actinomycosis, 531
nigricans, 531
- Striae, 222
Haab's, 296
hyaline, 416, 422
in keratoconus, 355, 414

- Striate keratitis, 421
 Stroma, corneal, 413
 alterations in, 413
 changes in thickness of, 414
 clefts in, 413
 deep inflammation of, 492
 edema of, 414
 fissures in, 414
 infiltration of, 414
 lines in, 413
 scar formation in, 415
 vascularization of, 415
 Swimming-bath conjunctivitis, 200
 Symblepharon, of conjunctiva, 155
 in pemphigus, 210, 222, 230
 Sympathetic ophthalmitis, keratitic pre-
 cipitates in, 426
 mutton-fat precipitates in, 433
 Synchysis scintillans, 594
 direct focal illumination of, 594
 Synechiae, congenital anterior, 569, 572
 postoperative, 665, 668
 prevention of, 650
 Syphilids, of conjunctiva, 219
 Syphilis, of conjunctiva, 215
 chancre in, 215
 conjunctivitis papulosa in, 215
 gumma in, 219
 panophthalmitis in, 219
 pseudotrachoma in, 215
 syphilids in, 219
 conjunctivitis, granular, 215
 of cornea, 503
 Parinaud's ocular glandular syndrome
 in, 221
 Tear film line, optic section in, 137
 Tears, in Bowman's membrane, 412
 in Descemet's membrane, 422
 Technique, adjustment of instrument,
 65
 of biomicroscopy, 64. (*See also* Bio-
 microscopy)
 fixation, 66
 illumination of room, 64
 preliminary survey of eyeball, 64
 preparation of patient, 65
 Telescope, binocular, 29
 Terminology, 11
 Thermal convection currents, 565
 keratic precipitates in, 433
 turbidity of aqueous in, 588
 Thermal injuries, of cornea, 555
 Trachoma, 202
 biomicroscopy of, 194, 202
 cicatricial tissue in, 207
 classification of, 202
 trachoma I, 202
 trachoma II, 202
 trachoma III, 207
 trachoma IV, 207
 complications of, 210
 differentiation of, from follicular con-
 junctivitis, 135, 194
 entropion in, 210
 facets in, 212
 follicles in, 184, 194, 202, 203
 Herbert's marginal pits in, 212
 hypertrophy in, 202
 madarosis in, 210
 optic section in, 202, 203
 pannus in, 210
 papillae in, 202
 pigmentation in, 370
 ptosis in, 210
 scars in, 207
 line of Arlt in, 207
 symblepharon in, 210
 trichiasis in, 210
 vascularization in, 203
 vital staining in, 203
 xerosis in, 210
 Transillumination, of conjunctiva, 136.
 (*See also* Retro-illumination)
 Transparent media, 9
 biomicroscopy of, 11
 Trauma. (*See also* Injuries)
 of anterior chamber, 572
 Trephining, 660
 Trichiasis, 210
 in blepharitis, 260
 Tubercle, of conjunctiva, 213
 miliary ulcers in, 214
 nodular type of, 214
 papillary hypertrophy in, 214
 polypoid type of, 214
 tuberculoma in, 214
 of cornea, 508
 Tuberculides, of conjunctiva, 214
 lichen scrofulosus type of, 214
 Tuberculoma, of conjunctiva, 214

- Tuberculosis, 508
 of cornea, 508
 scleroperikeratitis in, 513
 sclerocorneal, 511
 Tularemia, oculoglandular, 220
 conjunctivitis in, 220
 Tumors, of conjunctiva, 231
 angioma, 248
 congenital, 155
 nevus, 242
 cysts, 235
 epithelial, 231
 epithelioma, 236
 fibromas, 251
 granulomatous, 251
 hemangioma, 248
 nevus flammeus, 249
 leukosarcoma, 240
 lymphangiectases, 250
 lymphangioma, 250
 lymphatic, 236
 lymphoma, 250
 melanoma, malignant, 240
 melanosarcoma, 248
 mesoblastic, 251
 neuro-ectodermal, 240
 nevus, 242
 malignant, 243, 247
 papilloma, 231
 polyps, 251
 retention cysts, 235
 of vascular origin, 248
 of cornea, 231
 epithelioma, 236
 of eyelid margin, 265
 carcinoma, squamous-cell, 265
 epithelioma, basal cell, 265
 nevi, 265
 ulcer, rodent, 265
 lymphatic, 248
 mesoblastic, 251
 vascular, 248
 Turk's line, 371
 keratic precipitates in, 434
 Tylosis, 261
 Tyndall phenomenon, 10, 11
 in acute conjunctivitis, 185
 in anterior chamber, 563
 in keratic precipitates, 427
 in normal aqueous, 560
 Tyndallimetry, 588
 Ulbrich micrometer, 119
 in measuring depth of anterior chamber, 559
 Ulcer, atheromatous, 530
 of cornea, 520
 catarrhal, 522, 529
 folds in Descemet's membrane in, 417
 keratomycosis in, 531
 marginal, 525
 Morax-Axenfeld, 522
 Mooren's, 529
 dendritic, in herpes simplex corneae, 462, 463
 keratitis metaherpetica in, 467
 marginal, 525
 pneumococcal, 522
 rodent, 529
 of eyelid margin, 265
 serpiginous, 522
 chronic, 529
 ulcus corneae rodens, 529
 vascularization in, 403
 Ulcus serpens, 523
 Ultramicroscope, 10
 Universal slit lamp, 38
 Koeppel model, 37
 illumination for, 38, 40
 simplified model, 38
 Uvea, inflammation of, keratic precipitates in, 426
 Vaccination, keratitis from, 468
 Vacuole, of cornea, optic section, 109, 399
 of lens, optic section, 109
 retro-illumination of, 90
 unreversed illumination of, 399
 Varicella, 468
 Vascularization, in acne rosacea keratitis, 477
 of conjunctiva, 126
 bulbar vessels, 126
 limbus, 127
 tarsal vessels, 126
 of cornea, 401, 413
 superficial, 403
 in interstitial keratitis, 500
 in corneal scars, 536
 deep, 403
 in interstitial keratitis, 500
 direct focal illumination of, 502

- Vascularization— (*Continued*)
 in keratitis, interstitial, 500
 in pannus, 403
 superficial, 402
 Vasoconstrictors, 134
 Vasodilators, 134
 Vernal catarrh, 192
 corneal changes in, 449
 Vernal conjunctivitis, 192
 Verticillium graphii, 531
 Vesicular keratitis, 488
 Vesicles, epithelial, in diffuse vesicular keratitis, 488
 Vessels, conjunctival, circulation of, 132
 biomicroscopy of, 140
 corneal, direct focal illumination of, 409
 retro-illumination of, 408
 in interstitial keratitis, 502
 neoformation of, 402
 Virus, chickenpox, 468
 in inclusion conjunctivitis, 200
 infection, in inclusion blennorrhea, 200
 lymphogranuloma, 221
 neurotrophic, in herpes zoster keratitis, 466
 Visual palpation, 122
 Vital staining, aniline dyes in, 148
 anesthesia of conjunctiva in, 148
 azur ii in, 149
 Bengal rose in, 137
 of conjunctiva, 137
 method of, 148
 cresyl blue in, 149
 fluorescein in, 137, 149
 of cornea, 286
 abrasion of, 288
 aqueous solution in, 286, 287
 azur ii in, 286, 288
 caution in use of, 287
 cells of, 288
 epithelium of, desquamated epithelial cells in, 287
 fluorescein in, 289
 mercurochrome in, 286
 methylene blue in, 286, 288
 limbus, 289
 mucus in, 287
 nerves of, 289
 nile blue sulphate in, 288
 Vital staining— (*Continued*)
 of cornea— (*Continued*)
 of parenchymal cells, 288
 osmic acid solution in, 289
 polychrome methylene blue in, 286, 288
 rose Bengal in, 286, 288
 sodium fluorescein in, 286
 of corneal nerves, 443
 in keratitis, herpes zoster, 466
 in keratoconjunctivitis sicca, 471
 of lacrimal puncta, 269
 of lattice-like dystrophy, 338
 of lymphatics, 148
 hydrogen peroxide in, 148
 dionine in, 148
 cresyl brilliant blue in, 148
 of papillae, 178
 of pinguecula, 165
 in trachoma, 203
 Vitamin A deficiency, Bitot's spots in, 167
 Vitiligo, 173
 Vitreous, axis of beam in, 2
 illumination of, 66
 of observation in, 2, 66
 deep, examination of, 50
 direct focal illumination of, 78
 hand slit lamp in examining, 58
 herniations of, 585
 illuminating lens of 10 cm. in examining, 23
 prolapse of, into anterior chamber, 581
 Vogt method, of illumination, 8, 20.
 (*See also* Kohler-Vogt method)
 beam in, 24
 and Gullstrand method, 25
 Koeppé's tube in, 21
 plano-convex cylinder in, 21
 Vossius' ring, 414
 Wappler U-shaped hair-line filament, 58
 Warts, Hassall-Henle, on cornea, 279, 305
 War gases, 556
 White limbus girdle, 317, 366
 White rings of Coats, 316, 340
 etiology of, 340
 optic section of, 340
 Whorl opacity of cornea, 371



- Wilson's disease, 385
Wolff ophthalmoscope, 5
Wounds of cornea, 547
 perforating, 550

Xanthomatous patches, 165
Xanthomatosis of cornea, 352
Xerosis, of conjunctiva, 166, 210, 370
 corneae, 483
 differentiation of, from keratoconjunctivitis sicca, 470
 in keratitis e lagophthalmos, 476

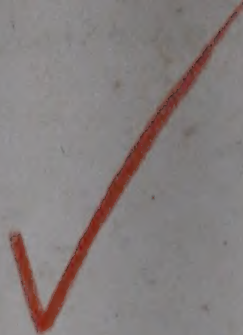
Xerosis—(*Continued*)
 prexerosis, 483

Zones of discontinuity, direct focal illumination in, 76
 of lens, 23, 103
 optical phenomena in, 94
 specular reflection in, 69
 corneal surface in, 97
 examination by, 91
 in edema of epithelium, 395
 localization in, 121

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